```
=> fil reg
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         0.21       0.21

FILE 'REGISTRY' ENTERED AT 12:14:54 ON 04 AUG 2006
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DICTIONARY FILE UPDATES:   2 AUG 2006  HIGHEST RN 898176-03-9

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predicted properties as well as tags indicating availability of
experimental property data in the original document.  For information
on property searching in REGISTRY, refer to:

http://www.cas.org/ONLINE/UG/regprops.html

=> e pramipexole/cn 5
E1            1     PRAMINIL/CN
E2            1     PRAMINO/CN
E3            1 --> PRAMIPEXOLE/CN
E4            1     PRAMIPEXOLE DIHYDROCHLORIDE/CN
E5            1     PRAMIPEXOLE DIHYDROCHLORIDE MONOHYDRATE/CN

=> s pramipexole ?/cn
L1            2 PRAMIPEXOLE ?/CN

=> e
E6            1     PRAMIPEXOLE HYDROCHLORIDE/CN
E7            1     PRAMIRACETAM/CN
E8            1     PRAMIRACETAM HYDROCHLORIDE/CN
E9            1     PRAMIRACETAM SULFATE/CN
E10           1     PRAMITOL/CN
E11           1     PRAMITOL 5P/CN
E12           1     PRAMIVERIN/CN
E13           1     PRAMIVERIN HYDROCHLORIDE/CN
E14           1     PRAMIVERINE/CN
E15           1     PRAMLINTIDE/CN
E16           1     PRAMLINTIDE ACETATE/CN
E17           1     PRAMLINTIDE ACETATE HYDRATE/CN

=> s e3-e6
              1 PRAMIPEXOLE/CN
              1 "PRAMIPEXOLE DIHYDROCHLORIDE"/CN
              1 "PRAMIPEXOLE DIHYDROCHLORIDE MONOHYDRATE"/CN
              1 "PRAMIPEXOLE HYDROCHLORIDE"/CN
L2            3 (PRAMIPEXOLE/CN OR "PRAMIPEXOLE DIHYDROCHLORIDE"/CN OR "PRAMIPEX

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```

OLE DIHYDROCHLORIDE MONOHYDRATE"/CN OR "PRAMIPEXOLE HYDROCHLORID
E"/CN)

```
=> s l1 or l2
L3            3 L1 OR L2

=> e type 2 diabetes/cn 5
E1            1     TYPE 1510/CN
E2            1     TYPE 18 SR/CN
E3            0 --> TYPE 2 DIABETES/CN
E4            1     TYPE 2 HELPER T-LYMPHOCYTE SPECIFIC PROTEIN 242C (MUS MUSCUL
                    US PRECURSOR)/CN
E5            1     TYPE 2 INOSITOL 1,4,5-TRISPHOSPHATE RECEPTOR (HUMAN CELL LIN
                    E NAMALWA)/CN

=> e diabetes type 2/cn 5
E1            1     DIABETAMID/CN
E2            1     DIABETES MELLITUS TYPE I AUTOANTIGEN (HUMAN CLONE RP11-505D1
                    7 GENE ICA1)/CN
E3            0 --> DIABETES TYPE 2/CN
E4            1     DIABETES-ASSOCIATED PEPTIDE/CN
E5            1     DIABETES-ASSOCIATED PEPTIDE (HUMAN)/CN

=> e diabetes mellitus type ii ?/cn
E1            1     DIABETAMID/CN
E2            1     DIABETES MELLITUS TYPE I AUTOANTIGEN (HUMAN CLONE RP11-505D1
                    7 GENE ICA1)/CN
E3            0 --> DIABETES MELLITUS TYPE II ?/CN
E4            1     DIABETES-ASSOCIATED PEPTIDE/CN
E5            1     DIABETES-ASSOCIATED PEPTIDE (HUMAN)/CN
E6            1     DIABETES-RELATED ANKYRIN REPEAT PROTEIN (HUMAN MUSCLE GENE D
                    ARP)/CN
E7            1     DIABETES-RELATED ANKYRIN REPEAT PROTEIN (MOUSE STRAIN C57BL/
                    6J GENE DARP)/CN
E8            1     DIABETIN/CN
E9            1     DIABETMIN/CN
E10           1     DIABETOGENIC FACTOR/CN
E11           1     DIABETOL/CN
E12           1     DIABETON/CN

=> => fil medl,biosis,embase
COST IN U.S. DOLLARS                          SINCE FILE      TOTAL
                                                   ENTRY    SESSION
FULL ESTIMATED COST                                3.90      29.67

FILE 'MEDLINE' ENTERED AT 12:22:46 ON 04 AUG 2006

FILE 'BIOSIS' ENTERED AT 12:22:46 ON 04 AUG 2006
Copyright (c) 2006 The Thomson Corporation

FILE 'EMBASE' ENTERED AT 12:22:46 ON 04 AUG 2006
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=> s (reduc? or decreas? or low?)(1)((food consump? or diet? or eat? behavior) or
over eating or food habit or feed? behavior? or food(w)(prefere? or intake) or
appetite)
L4       145481 FILE MEDLINE
L5       168903 FILE BIOSIS
L6       126283 FILE EMBASE
```

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TOTAL FOR ALL FILES
L7 440667 (REDUC? OR DECREAS? OR LOW?)(L)((FOOD CONSUMP? OR DIET? OR EAT? BEHAVIOR) OR OVER EATING OR FOOD HABIT OR FEED? BEHAVIOR? OR FOOD(W)(PREFERE? OR INTAKE) OR APPETITE)

=> s l1 or ?pramipexole?
L8 342 FILE MEDLINE
L9 473 FILE BIOSIS
L10 1592 FILE EMBASE

TOTAL FOR ALL FILES
L11 2407 L1 OR ?PRAMIPEXOLE?

=> s l7 and l11
L12 1 FILE MEDLINE
L13 2 FILE BIOSIS
L14 6 FILE EMBASE

TOTAL FOR ALL FILES
L15 9 L7 AND L11

=> dup rem l15
PROCESSING COMPLETED FOR L15
L16 7 DUP REM L15 (2 DUPLICATES REMOVED)

=> d 1-7 ibib abs

L16 ANSWER 1 OF 7 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2005231175 EMBASE
TITLE: Behavioral disturbances, not cognitive deterioration, are associated with altered food selection in seniors with Alzheimer's disease.
AUTHOR: Greenwood C.E.; Tam C.; Chan M.; Young K.W.H.; Binns M.A.; Van Reekum R.
CORPORATE SOURCE: Dr. C.E. Greenwood, Department of Nutritional Sciences, Faculty of Medicine, University of Toronto, Toronto, Ont. M5S 3E2, Canada. carol.greenwood@utoronto.ca
SOURCE: Journals of Gerontology - Series A Biological Sciences and Medical Sciences, (2005) Vol. 60, No. 4, pp. 499-505. .
Refs: 31
ISSN: 1079-5006 CODEN: JGASFW
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 008 Neurology and Neurosurgery
020 Gerontology and Geriatrics
029 Clinical Biochemistry
032 Psychiatry
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 16 Jun 2005
Last Updated on STN: 16 Jun 2005
AB Objective. We previously reported alterations in circadian patterns of food intake that are associated with measures of functional and cognitive deterioration in seniors with probable Alzheimer's disease (AD). This study further explored disturbed eating patterns in AD, focusing on alterations in macronutrient (protein, carbohydrate, and fat) selection, and their association with measures of functional and behavioral losses. Methods. Forty-nine days of food intake collections were conducted on 32 residents

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(26 females, 6 males; age = 88.4 ± 4.1 years; body mass index = 24.1 ± 4.0 kg/m (2)) with probable AD residing at a nursing home (a fully accredited geriatric teaching facility affiliated with the University of Toronto's Medical School). All residents ate their meals independently. The relationships between patterns of habitual food consumption and measures of cognitive function (Severe Impairment Battery), behavioral disturbances (Neuropsychiatric Inventory-Nursing Home Version) and behavioral function (London Psychogeriatric Rating Scale) were examined, cross-sectionally. Results. Consistent with our previous studies, breakfast intakes were not predicted by any of the measures of behavioral, cognitive, or functional deterioration, although those residents with greater functional deterioration, especially disengagement, attained lower 24-hour energy intakes. The presence of "psychomotor disturbances," including irritability, agitation, and disinhibition, were strongly associated with shifts in eating patterns toward carbohydrate and away from protein, placing individuals with these conditions at increased risk for inadequate protein intakes. Between-individual differences in intake patterns could not be explained by the use of either anorexic or orexigenic medications. Conclusions. Behavioral, not cognitive, deterioration is associated with appetite modifications that increase risk of poor protein intake, perhaps indicating a common monoaminergic involvement.

L16 ANSWER 2 OF 7 BIOSIS
ACCESSION NUMBER: 2004:401815 BIOSIS
DOCUMENT NUMBER: PREV200400401478
TITLE: Calming restless legs - Comment on Allen R et al. Ropinirole decreases periodic leg movements and improves sleep parameters in patients with restless legs syndrome. SLEEP 2004;27(5):907-14.
AUTHOR(S): Silber, Michael H. [Reprint Author]
CORPORATE SOURCE: Dept NeurolColl Med, Mayo Clin, 200 1st St SW, Rochester, MN, 55905, USA
msilber@mayo.edu
SOURCE: Sleep (Rochester), (August 1 2004) Vol. 27, No. 5, pp. 839-841. print.
CODEN: SLEED6. ISSN: 0161-8105.
DOCUMENT TYPE: Article
Editorial
LANGUAGE: English
ENTRY DATE: Entered STN: 13 Oct 2004
Last Updated on STN: 13 Oct 2004

L16 ANSWER 3 OF 7 EMBASE
ACCESSION NUMBER: 2001041233 EMBASE
TITLE: Parkinson's disease as multifactorial oxidative neurodegeneration: Implications for integrative management.
AUTHOR: Kidd P.M.
CORPORATE SOURCE: Dr. P.M. Kidd, Cell biology, University of California at Berkeley, 847 Elm St., El Cerrito, CA 94530, United States
SOURCE: Alternative Medicine Review, (2000) Vol. 5, No. 6, pp. 502-529. .
Refs: 123
ISSN: 1089-5159 CODEN: ALMRFP
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
008 Neurology and Neurosurgery

Prepared by: Mary Hale @2-2507 Rem Bldg 1D86

037 Drug Literature Index
038 Adverse Reactions Titles
052 Toxicology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 15 Feb 2001
Last Updated on STN: 15 Feb 2001

AB Parkinson's disease (PD) is the most common movement pathology, severely afflicting dopaminergic neurons within the substantia nigra (SN) along with non-dopaminergic, extra-nigral projection bundles that control circuits for sensory, associative, premotor, and motor pathways. Clinical, experimental, microanatomic, and biochemical evidence suggests PD involves multifactorial, oxidative neurodegeneration, and that levodopa therapy adds to the oxidative burden. The SN is uniquely vulnerable to oxidative damage, having a high content of oxidizable dopamine, neuromelanin, polyunsaturated fatty acids, and iron, and relatively low antioxidant complement with high metabolic rate. Oxidative phosphorylation abnormalities impair energetics in the SN mitochondria, also intensifying oxygen free radical generation. These pro-oxidative factors combine within the SN dopaminergic neurons to create extreme vulnerability to oxidative challenge. Epidemiologic studies and long-term tracking of victims of MPTP (1-methyl-4-phenyl-1,2,3,6,-tetrahydropyridine) poisoning, suggest oxidative stress compounded by exogenous toxins may trigger the neurodegenerative progression of PD. Rational, integrative management of PD requires: (1) dietary revision, especially to lower calories; (2) rebalancing of essential fatty acid intake away from pro-inflammatory and toward anti-inflammatory prostaglandins; (3) aggressive repletion of glutathione and other nutrient antioxidants and cofactors; (4) energy nutrients acetyl L-carnitine, coenzyme Q10, NADH, and the membrane phospholipid phosphatidylserine (PS); (5) chelation as necessary for heavy metals; and (6) liver P450 detoxification support.

L16 ANSWER 4 OF 7 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 1998070136 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9408198
TITLE: Differential behavioral responses to dopaminergic stimulation of nucleus accumbens subregions in the rat.
AUTHOR: Swanson C J; Heath S; Stratford T R; Kelley A E
CORPORATE SOURCE: Department of Psychiatry, University of Wisconsin-Madison Medical School, 53706, USA.
CONTRACT NUMBER: DA04788 (NIDA)
SOURCE: Pharmacology, biochemistry, and behavior, (1997 Dec) Vol. 58, No. 4, pp. 933-45.
Journal code: 0367050. ISSN: 0091-3057.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199801
ENTRY DATE: Entered STN: 17 Feb 1998
Last Updated on STN: 17 Feb 1998
Entered Medline: 30 Jan 1998

AB The following experiments investigated the behavioral response to local microinfusion of dopamine (DA) and selective DA agonists into the core and shell subregions of the nucleus accumbens. Rats were implanted with chronic indwelling cannulae aimed at these subregions. Two experiments were conducted. In experiment 1, the response to DA (0, 2, 5, 10 microg/0.5 microl/side), the D-1 agonist SKF-82598 (0, 0.1, 1.0 microg), the D-2/3 agonist quinpirole (0, 1, 5, 15 microg) and the D-3 preferring

agonist pramipexole (0.1, 1.0, 10.0 microg) was examined in photocell activity cages. Locomotor (horizontal) and rearing (vertical) activities were measured. DA and SKF-82958 induced relatively greater increases in activity following stimulation of the shell as compared with the core. Quinpirole induced a dose-dependent suppression of activity after infusion into both sites, although the core was more sensitive to the suppressive effect than the shell. Pramipexole induced time-dependent, biphasic effects that were small in magnitude and did not differentiate between site. In experiment 2, an observation procedure was used to record behaviors (locomotion, rearing, feeding, drinking). Dopamine (0, 2, 10 microg) elicited greater increases in rearing and feeding behavior in the shell than in the core. SKF-82958 (0, 0.75 microg) enhanced locomotion and rearing to a similar extent in both subregions in this test, whereas a mixture of a low dose (0.25 microg) of the D-1 and D-2 agonists selectively induced behavioral activation in the shell. In contrast to the results in the activity cage test, quinpirole (0, 1, 5 microg) increased motor activity at the lower dose when infused into the shell but not into the core. No alterations in feeding were observed following infusion of selective agonists, and no changes in drinking were found with any of the treatments. In summary, the shell appears to be relatively more sensitive to the motor activating effects of DA agonists than the core. Moreover, circuits associated with shell may be preferentially involved in feeding.

L16 ANSWER 5 OF 7 EMBASE

ACCESSION NUMBER: 95273297 EMBASE
DOCUMENT NUMBER: 1995273297
TITLE: Synthesis, pharmacological investigation and computational studies on a tricyclic ergoline analog with selective dopamine autoreceptor activity.
AUTHOR: Gmeiner P.; Bollinger B.; Mierau J.; Hofner G.
CORPORATE SOURCE: Pharmazeutisches Institut, Universitat Bonn, An der Immenburg 4,D-53121 Bonn, Germany
SOURCE: Archiv der Pharmazie, (1995) Vol. 328, No. 7-8, pp. 609-614. .
ISSN: 0365-6233 CODEN: ARPMAS
COUNTRY: Germany
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 008 Neurology and Neurosurgery
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 10 Oct 1995
Last Updated on STN: 10 Oct 1995

AB The novel aminobenzindolone 8 was prepared and evaluated as a potential antipsychotic agent. The target compound was synthesized in eight steps starting from the tetrahydrobenzindolone 9. The key step of the synthesis was an electrophilic amination of the aromatic ketone 11 followed by reductive degradation when the diethoxymethyl group was employed for protection of the lactam nitrogen and also for the benzylic position 2a. Dopamine and serotonin receptor binding studies revealed 8 to be a potent and selective ligand at the D-2 autoreceptor (k(i) = 4.0 nM). Further in vivo studies including the GBL-test and locomotor activity measurements indicated agonistic activity of 8 at the prejunctional binding sites. Comparison of ab initio based molecular electrostatic isopotential maps corroborates our hypothesis that the dopamine structure 6, containing an intramolecular hydrogen bond donating effect of the meta-HO-group, represents the conformation which is active

at the dopamine D-2 autoreceptor.

L16 ANSWER 6 OF 7 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 95044739 EMBASE
DOCUMENT NUMBER: 1995044739
TITLE: Dopamine-1 receptors in the proximal convoluted tubule of Dahl rats: Defective coupling to adenylate cyclase.
AUTHOR: Ohbu K.; Kaskel F.J.; Kinoshita S.; Felder R.A.
CORPORATE SOURCE: Dept. of Pathology, Virginia Univ. Health Sciences Ctr., Box 168,Charlottesville, VA 22908, United States
SOURCE: American Journal of Physiology - Regulatory Integrative and Comparative Physiology, (1995) Vol. 268, No. 1 37-1, pp. R231-R235. .
ISSN: 0363-6119 CODEN: AJPRDO
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 002 Physiology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 1 Mar 1995
Last Updated on STN: 1 Mar 1995

AB We have previously reported a defect in the coupling of the renal dopamine-1 receptor (D1) to adenylate cyclase (AC) in the proximal convoluted tubule (PCT) of the spontaneously hypertensive rat (Okamoto-Aoki strain). To determine if this defect is present in another model of hypertension, we microdissected PCTs from Dahl salt-sensitive (DSS) and Dahl salt-resistant (DSR) rats on low- or high-NaCl diet. The ability of two selective D1 agonists, fenoldopam and SND-919-C12, and forskolin to stimulate AC activity in PCT was determined in each of the four groups of rats. Fenoldopam (10-7 M) and SND-919-C12 (10-6 M) failed to stimulate AC activity in the PCT of DSS rats whether on a low- or high-NaCl diet. In DSR rats, however, both fenoldopam and SND-919-C12 stimulated AC activity by 289- 320% and 220-270%, respectively, whether on a low- or high-NaCl intake. Forskolin (10-5 M), which directly stimulates AC activity, increased AC activity in all four groups. These studies show that in DSS rats the D1 receptor in the PCT fails to respond to D1 agonists. This defect is not a consequence of the hypertension because it was present in the DSS rats on a low-salt diet and before blood pressure elevation.

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ACCESSION NUMBER: 94338342 EMBASE
DOCUMENT NUMBER: 1994338342
TITLE: Effect on rat feeding behavior of two selective D2 dopamine agonists.
AUTHOR: Ferrari F.; Guiliani D.
CORPORATE SOURCE: Department of Biomedical Sciences, Division of Pharmacology, University of Modena,41100 Modena, Italy
SOURCE: Physiology and Behavior, (1994) Vol. 56, No. 5, pp. 921-926. .
ISSN: 0031-9384 CODEN: PHBHA4
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 002 Physiology
003 Endocrinology
037 Drug Literature Index
LANGUAGE: English

Prepared by: Mary Hale @2-2507 Rem Bldg 1D86

SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 7 Dec 1994
Last Updated on STN: 7 Dec 1994
AB B-HT 958 and SND 919, two selective agonists at D2 dopamine receptors, were examined for their influence on the feeding behavior of fasted rats. When food intake was determined in the rat's individual home cage, it was found to be reduced by both drugs at low sedative doses during the first hour after treatment and by SND 919 at the highest dose (which also elicits stereotypy) only 24 h later. However, SND 919 and B-HT 958 had no significant effect on feeding evaluated according to the X-maze and tube feeding tests. Analysis of the results, seen in the context of other behavioral signs produced by the drugs, suggests that data on feeding may vary depending on the experimental model used and can be modified by extraneous factors that interfere with a specific effect on food intake.

=> s (obese or obesity or overweight or body mass or skin fold or body weight or overnutrition or nutrition disorder? or metabolic disease?)
L17 335586 FILE MEDLINE
L18 411500 FILE BIOSIS
L19 226099 FILE EMBASE

TOTAL FOR ALL FILES
L20 973185 (OBESE OR OBESITY OR OVERWEIGHT OR BODY MASS OR SKIN FOLD OR BODY WEIGHT OR OVERNUTRITION OR NUTRITION DISORDER? OR METABOLIC DISEASE?)

=> s l20 and l11
L21 3 FILE MEDLINE
L22 3 FILE BIOSIS
L23 25 FILE EMBASE

TOTAL FOR ALL FILES
L24 31 L20 AND L11

=> s l24 not l15
L25 3 FILE MEDLINE
L26 3 FILE BIOSIS
L27 24 FILE EMBASE

TOTAL FOR ALL FILES
L28 30 L24 NOT L15

=> dup rem l28
PROCESSING COMPLETED FOR L28
L29 27 DUP REM L28 (3 DUPLICATES REMOVED)

=> d 1-27 ibib abs hit

L29 ANSWER 1 OF 27 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2006325850 EMBASE
TITLE: Emerging pharmacological therapies for fibromyalgia.
AUTHOR: Lawson K.
CORPORATE SOURCE: K. Lawson, Biomedical Research Centre, Sheffield Hallam University, Faculty of Health and Wellbeing, City Campus, Sheffield S1 1WB, United Kingdom. K.Lawson@shu.ac.uk
SOURCE: Current Opinion in Investigational Drugs, (2006) Vol. 7,

No. 7, pp. 631-636. .
Refs: 58
ISSN: 1472-4472 CODEN: CIDREE
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 030 Pharmacology
031 Arthritis and Rheumatism
033 Orthopedic Surgery
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 1 Aug 2006
Last Updated on STN: 1 Aug 2006

AB Fibromyalgia is a chronic pain disorder for which pathophysiological mechanisms are difficult to identify and current drug therapies demonstrate limited effectiveness and significant tolerability. To date, no drugs have been officially approved for the indication of fibromyalgia, and randomized, controlled clinical trials with fibromyalgia patients are taking place to identify potential therapeutic approaches. Although emerging therapies, such as the antidepressants duloxetine and milnacipran and the antiepileptic pregabalin, offer certain efficacy, randomized controlled trials are generally difficult due to factors such as a lack of understanding of the pathophysiology and a heterogenous fibromyalgia patient population. For a significant advance in the drug treatment of fibromyalgia, novel clues are still awaited that may offer an effective therapeutic approach. .COPYRGT. The Thomson Corporation.

CT Medical Descriptors:
*fibromyalgia: DT, drug therapy
chronic pain: DT, drug therapy
pathophysiology
drug efficacy
drug tolerability
drug approval
drug indication
drug mechanism
nausea: SI, side effect
xerostomia: SI, side effect
constipation: SI, side effect
sweat gland disease: SI, side effect
abdominal pain: SI, side effect
drug induced headache: SI, side effect
dizziness: SI, side effect
flushing
side effect: SI, side effect
heart palpitation: SI, side effect
urine incontinence: SI, side effect
seizure: SI, side effect
coma: SI, side effect
drug fatality: SI, side effect
central nervous system depression
somnolence: SI, side effect
anxiety disorder: SI, side effect
weight reduction
body weight disorder: SI, side effect
human
clinical trial
review

CT Drug Descriptors:
duloxetine: AE, adverse drug reaction

Prepared by: Mary Hale @2-2507 Rem Bldg 1D86

duloxetine: CT, clinical trial
duloxetine: CM, drug comparison
duloxetine: DT, drug therapy
duloxetine: PD, pharmacology
milnacipran: AE, adverse drug reaction
milnacipran: CT, clinical trial
milnacipran: CM, drug comparison
milnacipran: DT, drug therapy
milnacipran: PD, pharmacology
pregabalin: AE, adverse drug reaction
pregabalin: CT, clinical trial
pregabalin: DT, drug therapy
pregabalin: PD, pharmacology
tricyclic antidepressant agent: AE, adverse drug reaction
tricyclic antidepressant agent: CM, drug comparison
tricyclic antidepressant agent: DT, drug therapy
tricyclic antidepressant agent: PD, pharmacology
serotonin uptake inhibitor: AE, adverse drug reaction
serotonin uptake inhibitor: CT, clinical trial
serotonin uptake inhibitor: DT, drug therapy
serotonin uptake inhibitor: PD, pharmacology
fluoxetine: DT, drug therapy
fluoxetine: PD, pharmacology
venlafaxine: CT, clinical trial
venlafaxine: DT, drug therapy
venlafaxine: PD, pharmacology
amitriptyline: AE, adverse drug reaction
amitriptyline: CM, drug comparison
amitriptyline: DT, drug therapy
amitriptyline: PD, pharmacology
moclobemide: CT, clinical trial
moclobemide: DT, drug therapy
moclobemide: PD, pharmacology
pargyline: CT, clinical trial
pargyline: DT, drug therapy
pargyline: PD, pharmacology
radafaxine: CT, clinical trial
radafaxine: DT, drug therapy
radafaxine: PD, pharmacology
noradrenalin uptake inhibitor: CT, clinical trial
noradrenalin uptake inhibitor: DT, drug therapy
noradrenalin uptake inhibitor: PD, pharmacology
ad 337: CT, clinical trial
ad 337: DT, drug therapy
ad 337: PD, pharmacology
zolpidem: DT, drug therapy
zolpidem: PD, pharmacology
zopiclone: DT, drug therapy
zopiclone: PD, pharmacology
eszopiclone: CT, clinical trial
eszopiclone: DT, drug therapy
eszopiclone: PD, pharmacology
indiplon: DT, drug therapy
indiplon: PD, pharmacology
gaboxadol: DT, drug therapy
gaboxadol: PD, pharmacology
hypnotic sedative agent: CT, clinical trial
hypnotic sedative agent: DT, drug therapy
hypnotic sedative agent: PD, pharmacology
oxybate sodium: AE, adverse drug reaction

oxybate sodium: CT, clinical trial
oxybate sodium: DT, drug therapy
oxybate sodium: PD, pharmacology
eplivanserin: CT, clinical trial
eplivanserin: DT, drug therapy
eplivanserin: PD, pharmacology
pramipexole: AE, adverse drug reaction
pramipexole: CT, clinical trial
pramipexole: DT, drug therapy
pramipexole: PD, pharmacology
ropinirole: CT, clinical trial
ropinirole: DT, drug therapy
ropinirole: PD, pharmacology
dronabinol: CT, clinical trial
dronabinol: DT, drug therapy
dronabinol: PD, pharmacology
hydrocortisone: CT, clinical trial
hydrocortisone: DT, drug therapy
hydrocortisone: PD, pharmacology
ibutamoren: CT, clinical trial
ibutamoren: DT, drug therapy
ibutamoren: PD, pharmacology
etiracetam: CT, clinical trial
etiracetam: DT, drug therapy
etiracetam: PD, pharmacology
modafinil: CT, clinical trial
modafinil: DT, drug therapy
modafinil: PD, pharmacology
zonisamide: CT, clinical trial
zonisamide: DT, drug therapy
zonisamide: PD, pharmacology
unindexed drug
unclassified drug

RN (duloxetine) 116539-59-4, 136434-34-9; (milnacipran) 101152-94-7, 86181-08-0, 92623-85-3; (pregabalin) 148553-50-8; (fluoxetine) 54910-89-3, 56296-78-7, 59333-67-4; (venlafaxine) 93413-69-5; (amitriptyline) 50-48-6, 549-18-8; (moclobemide) 71320-77-9; (pargyline) 306-07-0, 555-57-7; (radafaxine) 106083-71-0, 192374-14-4; (zolpidem) 82626-48-0; (zopiclone) 43200-80-2; (eszopiclone) 138729-47-2; (indiplon) 325715-02-4; (gaboxadol) 64603-91-4, 85118-33-8; (oxybate sodium) 502-85-2; (eplivanserin) 130580-02-8; (pramipexole) 104632-26-0; (ropinirole) 91374-21-9; (dronabinol) 7663-50-5; (hydrocortisone) 50-23-7; (ibutamoren) 159752-10-0; (etiracetam) 102767-28-2, 33996-58-6; (modafinil) 68693-11-8; (zonisamide) 68291-97-4

L29 ANSWER 2 OF 27 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2006228970 EMBASE
TITLE: Recent advances towards the discovery of dopamine receptor ligands.
AUTHOR: Zhang A.; Kan Y.; Li F.
CORPORATE SOURCE: Prof. A. Zhang, Synthetic Organic and Medicinal Chemistry Laboratory, Shanghai Institute of Materia Medica, Chinese Academy of Sciences, 555 Zuchongzhi Road, Zhangjiang Pudong, Shanghai 201203, China. aozhang@mail.shcnc.ac.cn
SOURCE: Expert Opinion on Therapeutic Patents, (2006) Vol. 16, No. 5, pp. 587-630. .
Refs: 103
ISSN: 1354-3776 CODEN: EOTPEG
COUNTRY: United Kingdom

Prepared by: Mary Hale @2-2507 Rem Bldg 1D86

DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 008 Neurology and Neurosurgery
030 Pharmacology
032 Psychiatry
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 1 Jun 2006
Last Updated on STN: 1 Jun 2006

AB Dopamine is a key regulator in the CNS, contributing importantly to functions of arousal and attention, initiation of movement, perception, motivation and emotion. Its imbalance has been implicated in the pathophysiology, and more clearly in the pharmacology, of a number of neurobehavioural disorders, including Parkinson's disease, schizophrenia, mania and depression, alcohol and drug abuse, as well as attention and eating disorders. Five major dopamine receptor subtypes (D1 - D5) have been identified, with distinct differences in their genes and peptide composition, molecular functions and neuropharmacology. These receptors represent the rational targets for the treatment of a large number of neurological and psychiatric disorders. In recent years, substantial efforts have addressed the most recently described dopamine receptor types, particularly types D3, D4 and D5, although most research involves the longer-known D1 and D2 dopamine receptors. Current pharmacological efforts in medicinal chemistry and neuropharmacology include the development of D1 full agonists and D2 partial agonists, as well as agents with dopaminergic activity combined with effects at CNS serotonergic, muscarinic, adrenergic and histaminic receptors. This review provides an overview of the recent patent literature during 2003-2005 on the development of therapeutic agents, mainly targeting the five dopamine receptors. .COPYRGT. 2006 Informa UK Ltd.

CT Medical Descriptors:
drug receptor binding
drug mechanism
Parkinson disease: DT, drug therapy
Parkinson disease: ET, etiology
neuroanatomy
substantia nigra
schizophrenia: DT, drug therapy
drug efficacy
drug structure
drug response
extrapyramidal symptom: SI, side effect
endocrine disease: SI, side effect
weight gain
body weight disorder: SI, side effect
receptor affinity
dystonia: SI, side effect
dyskinesia: SI, side effect
tardive dyskinesia: SI, side effect
drug bioavailability
drug metabolism
human
nonhuman
review

CT Drug Descriptors:
*dopamine receptor: EC, endogenous compound
*dopamine receptor stimulating agent: AE, adverse drug reaction
*dopamine receptor stimulating agent: AN, drug analysis
*dopamine receptor stimulating agent: CM, drug comparison

*dopamine receptor stimulating agent: DT, drug therapy
*dopamine receptor stimulating agent: PK, pharmacokinetics
*dopamine receptor stimulating agent: PD, pharmacology
*dopamine receptor stimulating agent: IP, intraperitoneal drug administration
*dopamine receptor stimulating agent: SC, subcutaneous drug administration
*dopamine receptor blocking agent: AE, adverse drug reaction
*dopamine receptor blocking agent: AN, drug analysis
*dopamine receptor blocking agent: CM, drug comparison
*dopamine receptor blocking agent: DT, drug therapy
*dopamine receptor blocking agent: PD, pharmacology
receptor subtype: EC, endogenous compound
dopamine 1 receptor: EC, endogenous compound
dopamine 2 receptor: EC, endogenous compound
dopamine 3 receptor: EC, endogenous compound
dopamine 4 receptor: EC, endogenous compound
dopamine 5 receptor: EC, endogenous compound
8 chloro 2,3,4,5 tetrahydro 3 methyl 5 phenyl 1h 3 benzazepin 7 ol hydrogen maleate: AN, drug analysis
8 chloro 2,3,4,5 tetrahydro 3 methyl 5 phenyl 1h 3 benzazepin 7 ol hydrogen maleate: PD, pharmacology
spiperone: PD, pharmacology
haloperidol: AE, adverse drug reaction
haloperidol: CM, drug comparison
haloperidol: DT, drug therapy
haloperidol: PD, pharmacology
ropinirole: DT, drug therapy
  pramipexole: DT, drug therapy
  pramipexole: PD, pharmacology
cabergoline: DT, drug therapy
chlorpromazine: AE, adverse drug reaction
chlorpromazine: CM, drug comparison
chlorpromazine: DT, drug therapy
chlorpromazine: PD, pharmacology
risperidone: AE, adverse drug reaction
risperidone: CM, drug comparison
risperidone: DT, drug therapy
risperidone: PD, pharmacology
levodopa: CM, drug comparison
levodopa: DT, drug therapy
levodopa: PD, pharmacology
apomorphine: AN, drug analysis
apomorphine: DT, drug therapy
apomorphine: PK, pharmacokinetics
apomorphine: PD, pharmacology
apomorphine: SC, subcutaneous drug administration
bromocriptine: DT, drug therapy
pergolide: DT, drug therapy
fluphenazine: AE, adverse drug reaction
fluphenazine: CM, drug comparison
fluphenazine: DT, drug therapy
fluphenazine: PD, pharmacology
clozapine: AE, adverse drug reaction
clozapine: AN, drug analysis
clozapine: CM, drug comparison
clozapine: DT, drug therapy
clozapine: PD, pharmacology
olanzapine: AE, adverse drug reaction
olanzapine: AN, drug analysis
olanzapine: CM, drug comparison

olanzapine: DT, drug therapy
olanzapine: PD, pharmacology
tiotixene: AE, adverse drug reaction
tiotixene: CM, drug comparison
tiotixene: DT, drug therapy
tiotixene: PD, pharmacology
trifluoperazine: AE, adverse drug reaction
trifluoperazine: CM, drug comparison
trifluoperazine: DT, drug therapy
trifluoperazine: PD, pharmacology
perphenazine: AE, adverse drug reaction
perphenazine: CM, drug comparison
perphenazine: DT, drug therapy
perphenazine: PD, pharmacology
quetiapine: AE, adverse drug reaction
quetiapine: AN, drug analysis
quetiapine: CM, drug comparison
quetiapine: DT, drug therapy
quetiapine: PD, pharmacology
aripiprazole: AE, adverse drug reaction
aripiprazole: AN, drug analysis
aripiprazole: CM, drug comparison
aripiprazole: DT, drug therapy
aripiprazole: PD, pharmacology
aripiprazole: IP, intraperitoneal drug administration
unindexed drug

RN (dopamine 4 receptor) 137750-34-6; (8 chloro 2,3,4,5 tetrahydro 3 methyl 5 phenyl 1h 3 benzazepin 7 ol hydrogen maleate) 87134-87-0; (spiperone) 749-02-0; (haloperidol) 52-86-8; (ropinirole) 91374-21-9; (pramipexole) 104632-26-0; (cabergoline) 81409-90-7; (chlorpromazine) 50-53-3, 69-09-0; (risperidone) 106266-06-2; (levodopa) 59-92-7; (apomorphine) 314-19-2, 58-00-4; (bromocriptine) 25614-03-3; (pergolide) 66104-22-1; (fluphenazine) 146-56-5, 69-23-8; (clozapine) 5786-21-0; (olanzapine) 132539-06-1; (tiotixene) 5591-45-7; (trifluoperazine) 117-89-5, 440-17-5; (perphenazine) 58-39-9; (quetiapine) 111974-72-2; (aripiprazole) 129722-12-9

L29 ANSWER 3 OF 27 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2006210889 IN-PROCESS
DOCUMENT NUMBER: PubMed ID: 16261618
TITLE: Compulsive eating and weight gain related to dopamine agonist use.
AUTHOR: Nirenberg Melissa J; Waters Cheryl
CORPORATE SOURCE: Division of Movement Disorders, Department of Neurology, Columbia University Medical Center, New York, NY 10021, USA.. mjnirenb@med.cornell.edu
SOURCE: Movement disorders : official journal of the Movement Disorder Society, (2006 Apr) Vol. 21, No. 4, pp. 524-9. Journal code: 8610688. ISSN: 0885-3185.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: NONMEDLINE; IN-PROCESS; NONINDEXED; Priority Journals
ENTRY DATE: Entered STN: 18 Apr 2006
Last Updated on STN: 10 May 2006

AB Dopamine agonists have been implicated in causing compulsive behaviors in patients with Parkinson's disease (PD). These have included gambling, hypersexuality, hobbyism, and other repetitive, purposeless behaviors ("punding"). In this report, we describe 7 patients in whom compulsive eating developed in the context of pramipexole use. All of the

affected patients had significant, undesired weight gain; 4 had other comorbid compulsive behaviors. In the 5 patients who lowered the dose of pramipexole or discontinued dopamine agonist treatment, the behavior remitted and no further weight gain occurred. Physicians should be aware that compulsive eating resulting in significant weight gain may occur in PD as a side-effect of dopamine agonist medications such as pramipexole. Given the known risks of the associated weight gain and obesity, further investigation is warranted.

AB Dopamine agonists have been implicated in causing compulsive behaviors in patients with Parkinson's disease (PD). These have included gambling, hypersexuality, hobbyism, and other repetitive, purposeless behaviors ("punding"). In this report, we describe 7 patients in whom compulsive eating developed in the context of pramipexole use. All of the affected patients had significant, undesired weight gain; 4 had other comorbid compulsive behaviors. In the 5 patients who lowered the dose of pramipexole or discontinued dopamine agonist treatment, the behavior remitted and no further weight gain occurred. Physicians should be aware that compulsive eating resulting in significant weight gain may occur in PD as a side-effect of dopamine agonist medications such as pramipexole. Given the known risks of the associated weight gain and obesity, further investigation is warranted.

L29 ANSWER 4 OF 27 EMBASE

ACCESSION NUMBER: 2006122094 EMBASE
TITLE: Pramipexole for the treatment of restless legs syndrome.
AUTHOR: Kushida C.A.
CORPORATE SOURCE: Dr. C.A. Kushida, Stanford University Center of Excellence for Sleep Disorders, 401 Quarry Road, Stanford, CA 94305-5730, United States. clete@stanford.edu
SOURCE: Expert Opinion on Pharmacotherapy, (2006) Vol. 7, No. 4, pp. 441-451. .
Refs: 64
ISSN: 1465-6566 CODEN: EOPHF7
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 008 Neurology and Neurosurgery
030 Pharmacology
033 Orthopedic Surgery
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 28 Mar 2006
Last Updated on STN: 28 Mar 2006

AB Restless legs syndrome (RLS) is a common disorder that is estimated to affect 10% of Americans. However, it remains largely undiagnosed and untreated by clinicians. The primary symptoms of this condition are leg discomfort or an urge to move that is temporarily relieved by movement and is worse at rest and at bedtime. RLS impacts the quality of life of the sufferer by disrupting sleep and disturbing or curtailing work and social activities. Approximately 80% of RLS sufferers also have periodic limb movements during sleep, in which repetitive leg movements fragment sleep and may result in daytime drowsiness. RLS may be treated by dopaminergic agents, benzodiazepines, anticonvulsants and opiates; dopamine agonists are currently considered first-line therapy for this condition. Pramipexole has been studied in the treatment of RLS since 1998.

Prepared by: Mary Hale @2-2507 Rem Bldg 1D86

This article reviews the role of this medication in the management of this serious neurological disorder. .COPYRGT. 2006 Ashley Publications.

TI Pramipexole for the treatment of restless legs syndrome.

AB Restless legs syndrome (RLS) is a common disorder that is estimated to affect 10% of Americans. However, it remains largely undiagnosed and untreated by clinicians. The primary symptoms of this condition are leg discomfort or an urge to move that is temporarily relieved by movement and is worse at rest and at bedtime. RLS impacts the quality of life of the sufferer by disrupting sleep and disturbing or curtailing work and social activities. Approximately 80% of RLS sufferers also have periodic limb movements during sleep, in which repetitive leg movements fragment sleep and may result in daytime drowsiness. RLS may be treated by dopaminergic agents, benzodiazepines, anticonvulsants and opiates; dopamine agonists are currently considered first-line therapy for this condition. Pramipexole has been studied in the treatment of RLS since 1998. This article reviews the role of this medication in the management of this serious neurological disorder. .COPYRGT. 2006 Ashley Publications.

CT Medical Descriptors:

*restless legs syndrome: DI, diagnosis
*restless legs syndrome: DT, drug therapy
*restless legs syndrome: EP, epidemiology
*restless legs syndrome: TH, therapy
incidence
leg movement
disease severity
rest
quality of life
sleep disorder: ET, etiology
sleep disorder: TH, therapy
social behavior
diagnostic procedure
polysomnography
exercise
psychophysiology
massage
acupuncture
drug efficacy
drug safety
drug tolerability
drug metabolism
drug dose regimen
insomnia: SI, side effect
nausea: SI, side effect
dyspepsia: SI, side effect
dizziness: SI, side effect
constipation: SI, side effect
fatigue: SI, side effect
anorexia: SI, side effect
somnolence: SI, side effect
rhinopharyngitis: SI, side effect
asthenia: SI, side effect
orthostatic hypotension: SI, side effect
dyskinesia: SI, side effect
body weight disorder: SI, side effect
hypersexuality: SI, side effect
human
clinical trial
review

CT Drug Descriptors:

*pramipexole: AE, adverse drug reaction

*pramipexole: CT, clinical trial
*pramipexole: DO, drug dose
*pramipexole: DT, drug therapy
*pramipexole: PK, pharmacokinetics
*pramipexole: PD, pharmacology
dopamine receptor stimulating agent: DT, drug therapy
benzodiazepine: DT, drug therapy
anticonvulsive agent: DT, drug therapy
opiate: DT, drug therapy
placebo
clonazepam: DT, drug therapy
levodopa: DT, drug therapy
pergolide: DT, drug therapy
RN (pramipexole) 104632-26-0; (benzodiazepine) 12794-10-4; (opiate) 53663-61-9, 8002-76-4, 8008-60-4; (clonazepam) 1622-61-3; (levodopa) 59-92-7; (pergolide) 66104-22-1

L29 ANSWER 5 OF 27 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2006312221 EMBASE
TITLE: Dopaminergic-based pharmacotherapies for depression.
AUTHOR: Papakostas G.I.
CORPORATE SOURCE: G.I. Papakostas, Depression Clinical and Research Program, Massachusetts General Hospital, Harvard Medical School, Boston, MA, United States. gpapakostas@partners.org
SOURCE: European Neuropsychopharmacology, (2006) Vol. 16, No. 6, pp. 391-402. .
Refs: 185
ISSN: 0924-977X CODEN: EURNE8
PUBLISHER IDENT.: S 0924-977X(05)00211-7
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 008 Neurology and Neurosurgery
032 Psychiatry
036 Health Policy, Economics and Management
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 1 Aug 2006
Last Updated on STN: 1 Aug 2006

AB The serendipitous discovery of the precursors of two of the major contemporary antidepressant families during the late 1950s, iproniazid for the monoamine oxidase inhibitors (MAOIs) and imipramine for the tricyclic antidepressants (TCAs), has guided the subsequent development of antidepressant compounds with predominantly serotonergic, noradrenergic or combined serotonergic and noradrenergic activity. Unfortunately, however, many depressed patients continue to remain symptomatic despite adequate treatment with pharmacologic agents currently available. When one reviews the list of pharmacologic agents currently approved for the treatment of Major Depressive Disorder (MDD), it is apparent that relatively few treatments with dopaminergic activity have been developed to date. Therefore, developing effective antidepressant treatments with pro-dopaminergic properties which also possess a relatively wide safety margin may further improve the standard of care for depression. In the present article we will briefly review studies focusing on the role of dopamine in depression followed by a comprehensive review of pharmacotherapies for depression with pro-dopaminergic activity. .COPYRGT. 2005 Elsevier B.V. and ECNP.

CT Medical Descriptors:

*major depression: DR, drug resistance
*major depression: DT, drug therapy
*major depression: ET, etiology
*dopaminergic system
*neuropharmacology
drug research
dopaminergic activity
serotonergic activity
noradrenergic activity
drug activity
drug safety
dopamine uptake
neuropathology
central nervous system
neurophysiology
dopamine metabolism
monoamine metabolism
drug mechanism
drug approval
fibromyalgia: DT, drug therapy
chronic fatigue syndrome: DT, drug therapy
Parkinson disease: DT, drug therapy
cognitive defect: DT, drug therapy
anxiety disorder: DT, drug therapy
psychomotor retardation: DT, drug therapy
  obesity: DT, drug therapy
dose response
hypertensive crisis: SI, side effect
serotonin syndrome: SI, side effect
diarrhea: SI, side effect
liver toxicity: SI, side effect
sexual dysfunction: SI, side effect
fatigue: SI, side effect
drug fever: SI, side effect
nausea: SI, side effect
gastrointestinal toxicity: SI, side effect
  body weight disorder: SI, side effect
seizure: SI, side effect
hemolytic anemia: SI, side effect
drug dependence
human
nonhuman
clinical trial
meta analysis
systematic review
review
priority journal
CT Drug Descriptors:
*antidepressant agent: AE, adverse drug reaction
*antidepressant agent: CT, clinical trial
*antidepressant agent: CM, drug comparison
*antidepressant agent: DO, drug dose
*antidepressant agent: DT, drug therapy
*antidepressant agent: TO, drug toxicity
*antidepressant agent: PE, pharmacoeconomics
*antidepressant agent: PD, pharmacology
phenelzine: PE, pharmacoeconomics
phenelzine: PD, pharmacology
tranylcypromine: PE, pharmacoeconomics
tranylcypromine: PD, pharmacology

isocarboxazid: PE, pharmacoeconomics
isocarboxazid: PD, pharmacology
imipramine: PD, pharmacology
brofaromine: PD, pharmacology
moclobemide: PD, pharmacology
selegiline: DO, drug dose
selegiline: PD, pharmacology
monoamine oxidase inhibitor: AE, adverse drug reaction
monoamine oxidase inhibitor: CT, clinical trial
monoamine oxidase inhibitor: CM, drug comparison
monoamine oxidase inhibitor: DO, drug dose
monoamine oxidase inhibitor: DT, drug therapy
monoamine oxidase inhibitor: PE, pharmacoeconomics
monoamine oxidase inhibitor: PD, pharmacology
iproniazid: PD, pharmacology
tricyclic antidepressant agent: CM, drug comparison
tricyclic antidepressant agent: PD, pharmacology
tolcapone: AE, adverse drug reaction
tolcapone: CT, clinical trial
tolcapone: DT, drug therapy
tolcapone: PE, pharmacoeconomics
tolcapone: PD, pharmacology
entacapone: PE, pharmacoeconomics
entacapone: PD, pharmacology
catecholamine o methyltransferase inhibitor: AE, adverse drug reaction
catecholamine o methyltransferase inhibitor: CT, clinical trial
catecholamine o methyltransferase inhibitor: DT, drug therapy
catecholamine o methyltransferase inhibitor: PE, pharmacoeconomics
catecholamine o methyltransferase inhibitor: PD, pharmacology
fluoxetine: AE, adverse drug reaction
fluoxetine: CT, clinical trial
fluoxetine: CM, drug comparison
fluoxetine: DT, drug therapy
sertraline: AE, adverse drug reaction
sertraline: CT, clinical trial
sertraline: CM, drug comparison
sertraline: DT, drug therapy
paroxetine: AE, adverse drug reaction
paroxetine: CT, clinical trial
paroxetine: CM, drug comparison
paroxetine: DT, drug therapy
escitalopram: AE, adverse drug reaction
escitalopram: CT, clinical trial
escitalopram: CM, drug comparison
escitalopram: DT, drug therapy
serotonin uptake inhibitor: AE, adverse drug reaction
serotonin uptake inhibitor: CT, clinical trial
serotonin uptake inhibitor: CM, drug comparison
serotonin uptake inhibitor: DO, drug dose
serotonin uptake inhibitor: DT, drug therapy
nomifensine maleate: AE, adverse drug reaction
nomifensine maleate: CM, drug comparison
nomifensine maleate: DT, drug therapy
nomifensine maleate: PD, pharmacology
amfebutamone: AE, adverse drug reaction
amfebutamone: CT, clinical trial
amfebutamone: CM, drug comparison
amfebutamone: DO, drug dose
amfebutamone: DT, drug therapy
amfebutamone: PD, pharmacology

sibutramine: CT, clinical trial
sibutramine: DT, drug therapy
sibutramine: PD, pharmacology
amineptine: AE, adverse drug reaction
amineptine: CT, clinical trial
amineptine: CM, drug comparison
amineptine: DT, drug therapy
amineptine: TO, drug toxicity
amineptine: PD, pharmacology
dopamine uptake inhibitor: AE, adverse drug reaction
dopamine uptake inhibitor: CT, clinical trial
dopamine uptake inhibitor: CM, drug comparison
dopamine uptake inhibitor: DO, drug dose
dopamine uptake inhibitor: DT, drug therapy
dopamine uptake inhibitor: TO, drug toxicity
dopamine uptake inhibitor: PD, pharmacology
pemoline: CT, clinical trial
pemoline: CM, drug comparison
pemoline: DT, drug therapy
pemoline: PD, pharmacology
dexamphetamine: CT, clinical trial
dexamphetamine: CM, drug comparison
dexamphetamine: DT, drug therapy
dexamphetamine: PD, pharmacology
methylphenidate: CT, clinical trial
methylphenidate: CM, drug comparison
methylphenidate: DT, drug therapy
methylphenidate: PD, pharmacology
dopamine receptor affecting agent: CT, clinical trial
dopamine receptor affecting agent: CB, drug combination
dopamine receptor affecting agent: CM, drug comparison
dopamine receptor affecting agent: DT, drug therapy
dopamine receptor affecting agent: PE, pharmacoeconomics
dopamine receptor affecting agent: PD, pharmacology
placebo
unindexed drug
unclassified drug
piribedil
bromocriptine mesilate
amantadine
pergolide mesilate
pramipexole
ropinirole

RN (phenelzine) 156-51-4, 51-71-8; (tranylcypromine) 13492-01-8, 155-09-9, 54-97-7; (isocarboxazid) 59-63-2; (imipramine) 113-52-0, 50-49-7; (brofaromine) 63638-90-4; (moclobemide) 71320-77-9; (selegiline) 14611-51-9, 14611-52-0, 2079-54-1, 2323-36-6; (iproniazid) 305-33-9, 54-92-2; (tolcapone) 134308-13-7; (entacapone) 116314-67-1; (fluoxetine) 54910-89-3, 56296-78-7, 59333-67-4; (sertraline) 79617-96-2; (paroxetine) 61869-08-7; (escitalopram) 128196-01-0, 219861-08-2; (nomifensine maleate) 32795-47-4; (amfebutamone) 31677-93-7, 34911-55-2; (sibutramine) 106650-56-0; (amineptine) 30272-08-3, 57574-09-1; (pemoline) 2152-34-3; (dexamphetamine) 1462-73-3, 51-63-8, 51-64-9; (methylphenidate) 113-45-1, 298-59-9; (piribedil) 3605-01-4; (bromocriptine mesilate) 22260-51-1; (amantadine) 665-66-7, 768-94-5; (pergolide mesilate) 66104-23-2; (pramipexole) 104632-26-0; (ropinirole) 91374-21-9

L29 ANSWER 6 OF 27 EMBASE
ACCESSION NUMBER: 2006202754 EMBASE

TITLE: Loosening addiction's deadly grip.
AUTHOR: Brower V.
SOURCE: EMBO Reports, (2006) Vol. 7, No. 2, pp. 140-142. .
Refs: 19
ISSN: 1469-221X E-ISSN: 1469-3178 CODEN: ERMEAX
PUBLISHER IDENT.: 7400635
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 008 Neurology and Neurosurgery
032 Psychiatry
037 Drug Literature Index
038 Adverse Reactions Titles
040 Drug Dependence, Alcohol Abuse and Alcoholism
LANGUAGE: English
ENTRY DATE: Entered STN: 25 May 2006
Last Updated on STN: 25 May 2006

DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

CT Medical Descriptors:
*drug dependence: DT, drug therapy
medical research
neurologic disease
Parkinson disease: DT, drug therapy
pathological gambling: SI, side effect
drug dose reduction
overnutrition: SI, side effect
hypersexuality: SI, side effect
social problem
public health
drug abuse
prescription
brain disease
reinforcement
attention deficit disorder: DT, drug therapy
alcohol consumption
anxiety
mental disease: DT, drug therapy
cocaine dependence: DT, drug therapy
alcoholism: DT, drug therapy
human
nonhuman
clinical trial
article
priority journal
CT Drug Descriptors:
pramipexole: AE, adverse drug reaction
pramipexole: DO, drug dose
pramipexole: DT, drug therapy
dopamine 3 receptor stimulating agent: AE, adverse drug reaction
dopamine 3 receptor stimulating agent: DO, drug dose
dopamine 3 receptor stimulating agent: DT, drug therapy
cocaine
methamphetamine
alcohol
illicit drug
opiate
central stimulant agent
central depressant agent
dopamine
nicotine
methylphenidate: DT, drug therapy

Prepared by: Mary Hale @2-2507 Rem Bldg 1D86

aripiprazole: DT, drug therapy
modafinil: DT, drug therapy
naltrexone: DT, drug therapy
talampanel: CT, clinical trial
talampanel: DT, drug therapy

RN (pramipexole) 104632-26-0; (cocaine) 50-36-2, 53-21-4, 5937-29-1; (methamphetamine) 28297-73-6, 51-57-0, 537-46-2, 7632-10-2; (alcohol) 64-17-5; (opiate) 53663-61-9, 8002-76-4, 8008-60-4; (dopamine) 51-61-6, 62-31-7; (nicotine) 54-11-5; (methylphenidate) 113-45-1, 298-59-9; (aripiprazole) 129722-12-9; (modafinil) 68693-11-8; (naltrexone) 16590-41-3, 16676-29-2; (talampanel) 161832-65-1, 161832-67-3

L29 ANSWER 7 OF 27 EMBASE

ACCESSION NUMBER: 2006023991 EMBASE
TITLE: Sleep apnea syndrome in Parkinson's disease. A case-control study in 49 patients.
AUTHOR: Diederich N.J.; Vaillant M.; Leischen M.; Mancuso G.; Golinval S.; Nati R.; Schlesser M.
CORPORATE SOURCE: Dr. N.J. Diederich, Department of Neuroscience, Centre Hospitalier de Luxembourg, 4 rue Barble, L-1210 Luxembourg, Luxembourg. diederdn@pt.lu
SOURCE: Movement Disorders, (2005) Vol. 20, No. 11, pp. 1413-1418.
Refs: 19
ISSN: 0885-3185 CODEN: MOVDEA
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 008 Neurology and Neurosurgery
015 Chest Diseases, Thoracic Surgery and Tuberculosis
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 2 Feb 2006
Last Updated on STN: 2 Feb 2006

AB In PD, the impact of nocturnal respiration on sleep continuity and architecture has not been systematically investigated by polysomnography (PSG). We performed a case-control study with retrospective analysis of PSG data of 49 PD patients. After classifying the PD patients according to their apnea/hypopnea index (AHI), they were matched with 49 controls in terms of age, gender, and AFR. There were 21 PD patients (43%) who had sleep apnea syndrome (SAS), classified as mild (AHI, 5-15) in 10 patients, moderate (AHI, >15-30) in 4 patients, and severe (AHI, > 30) in 7 patients. PD patients had more deep sleep (P = 0.02) and more nocturnal awakenings (P < 0.001) than the controls. Their body mass index (BMI) was lower (P = 0.04), and they maintained a more favorable respiratory profile, with higher mean and minimal oxygen saturation values (P = 0.006 and 0.01, respectively). These differences were preserved when only considering PD patients with AHI > 15. PD patients had less obstructive sleep apneas (P = 0.035), independently from the factor AHI. Only the respiratory changes of 4 PD patients with BMI > 27 and AHI > 15 (8%) approximated those seen in the controls. At an early or middle stage of the disease, non-obese PD patients frequently have AM values suggesting SAS, however, without the oxygen desaturation profile of SAS. Longitudinal studies of patients with such "abortive" SAS are warranted to establish if this finding reflects benign nocturnal respiratory muscle dyskinesia or constitutes a precursor sign of dysautonomia in PD. .COPYRGT. 2005 Movement Disorder Society.
AB In PD, the impact of nocturnal respiration on sleep continuity and architecture has not been systematically investigated by polysomnography

(PSG). We performed a case-control study with retrospective analysis of PSG data of 49 PD patients. After classifying the PD patients according to their apnea/hypopnea index (AHI), they were matched with 49 controls in terms of age, gender, and AFR. There were 21 PD patients (43%) who had sleep apnea syndrome (SAS), classified as mild (AHI, 5-15) in 10 patients, moderate (AHI, >15-30) in 4 patients, and severe (AHI, > 30) in 7 patients. PD patients had more deep sleep (P = 0.02) and more nocturnal awakenings (P < 0.001) than the controls. Their body mass index (BMI) was lower (P = 0.04), and they maintained a more favorable respiratory profile, with higher mean and minimal oxygen saturation values (P = 0.006 and 0.01, respectively). These differences were preserved when only considering PD patients with AHI > 15. PD patients had less obstructive sleep apneas (P = 0.035), independently from the factor AHI. Only the respiratory changes of 4 PD patients with BMI > 27 and AHI > 15 (8%) approximated those seen in the controls. At an early or middle stage of the disease, non-obese PD patients frequently have AM values suggesting SAS, however, without the oxygen desaturation profile of SAS. Longitudinal studies of patients with such "abortive" SAS are warranted to establish if this finding reflects benign nocturnal respiratory muscle dyskinesia or constitutes a precursor sign of dysautonomia in PD. .COPYRGT. 2005 Movement Disorder Society.

CT Medical Descriptors:
*Parkinson disease: DT, drug therapy
*sleep apnea syndrome
case control study
retrospective study
disease severity
REM sleep
wakefulness
body mass
oxygen saturation
dyskinesia
human
male
female
clinical article
controlled study
aged
adult
article
priority journal
Drug Descriptors:
levodopa: DT, drug therapy
dopamine receptor stimulating agent: DT, drug therapy
pergolide: DT, drug therapy
bromocriptine: DT, drug therapy
pramipexole: DT, drug therapy
ropinirole: DT, drug therapy

RN (levodopa) 59-92-7; (pergolide) 66104-22-1; (bromocriptine) 25614-03-3; (pramipexole) 104632-26-0; (ropinirole) 91374-21-9

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ACCESSION NUMBER: 2005204189 EMBASE
TITLE: Adjunctive strategies in the treatment of refractory bipolar depression: Clinician options in the absence of a systematic database.
AUTHOR: Post R.M.
CORPORATE SOURCE: Dr. R.M. Post, National Institute of Mental Health, Biological Psychiatry Branch, Department of Health and

Human Services, 10 Center Drive MSC 1272, Bldg. 10,
Bethesda, MD 20892-1272, United States. Robert.Post@nih.gov
SOURCE: Expert Opinion on Pharmacotherapy, (2005) Vol. 6, No. 4,
pp. 531-546. .
Refs: 140
ISSN: 1465-6566 CODEN: EOPHF7
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 032 Psychiatry
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 26 May 2005
Last Updated on STN: 26 May 2005

AB Multiple approaches to enhancing antidepressant treatment response in bipolar depression are available and should, in many instances, be explored despite a lack of definitive controlled trial literature supporting their efficacy. Given that the morbidity of depression is three times greater than mania in bipolar illness, a range of treatment approaches to this phase of illness should be pursued. This paper highlights the preliminary evidence of efficacy versus side effects, tolerability, and safety in order to suggest an overall provisional utility grade for each well-studied to highly-experimental option. Given the general paucity of evidence to support efficacy or to sequence different approaches for augmenting treatment of bipolar depression, it is critical that patient and physician adopt a systematic and, preferably, daily rating approach to the assessment of benefit for a given patient of each strategy contemplated. The goal is to achieve and maintain remission of depressive symptoms and associated comorbidities, which is often not accomplished using primary mood stabiliser treatments alone, or in combination; thus, an active clinical approach to augmentation strategies is indicated even when the literature provides only highly preliminary guidance.

CT Medical Descriptors:
*bipolar depression: DT, drug therapy
*bipolar depression: SU, surgery
*bipolar depression: TH, therapy
data base
drug response
drug efficacy
bipolar mania: DT, drug therapy
bipolar disorder: DT, drug therapy
side effect: SI, side effect
drug tolerability
drug safety
remission
comorbidity
agranulocytosis: SI, side effect
aplastic anemia: SI, side effect
spina bifida: SI, side effect
rash: SI, side effect
Stevens Johnson syndrome: SI, side effect
toxic epidermal necrolysis: SI, side effect
tremor: SI, side effect
gastrointestinal symptom: SI, side effect
weight gain
body weight disorder: SI, side effect
diabetes insipidus: SI, side effect
hyponatremia: SI, side effect

alopecia: SI, side effect
ovary polycystic disease: SI, side effect
acne: SI, side effect
psoriasis: SI, side effect
leukopenia: SI, side effect
dizziness: SI, side effect
ataxia: SI, side effect
diplopia: SI, side effect
sedation
somnolence: SI, side effect
thrombocytopenia: SI, side effect
headache: SI, side effect
insomnia: SI, side effect
orthostatic hypotension: SI, side effect
extrapyramidal symptom: SI, side effect
tardive dyskinesia: SI, side effect
akathisia: SI, side effect
drug potentiation
sexual dysfunction: SI, side effect
drug half life
drug megadose
drug targeting
mania: SI, side effect
hypotension: SI, side effect
xerostomia: SI, side effect
human
review

CT Drug Descriptors:
antidepressant agent: DT, drug therapy
mood stabilizer: DT, drug therapy
lithium: AE, adverse drug reaction
lithium: CB, drug combination
lithium: IT, drug interaction
lithium: DT, drug therapy
carbamazepine: AE, adverse drug reaction
carbamazepine: CB, drug combination
carbamazepine: DT, drug therapy
valproic acid: AE, adverse drug reaction
valproic acid: CB, drug combination
valproic acid: DT, drug therapy
lamotrigine: AE, adverse drug reaction
lamotrigine: CB, drug combination
lamotrigine: DT, drug therapy
neuroleptic agent: AE, adverse drug reaction
neuroleptic agent: DT, drug therapy
molindone: AE, adverse drug reaction
molindone: DT, drug therapy
atypical antipsychotic agent: AE, adverse drug reaction
atypical antipsychotic agent: DT, drug therapy
clozapine: AE, adverse drug reaction
clozapine: CM, drug comparison
clozapine: DT, drug therapy
risperidone: AE, adverse drug reaction
risperidone: CM, drug comparison
risperidone: DT, drug therapy
olanzapine: AE, adverse drug reaction
olanzapine: CB, drug combination
olanzapine: CM, drug comparison
olanzapine: DT, drug therapy
quetiapine: AE, adverse drug reaction

quetiapine: CM, drug comparison
quetiapine: DT, drug therapy
ziprasidone: AE, adverse drug reaction
ziprasidone: DT, drug therapy
aripiprazole: DT, drug therapy
tricyclic antidepressant agent: DT, drug therapy
imipramine: DT, drug therapy
amfebutamone: CB, drug combination
amfebutamone: CM, drug comparison
amfebutamone: DT, drug therapy
amfebutamone: PD, pharmacology
serotonin uptake inhibitor: AE, adverse drug reaction
serotonin uptake inhibitor: CB, drug combination
serotonin uptake inhibitor: CM, drug comparison
serotonin uptake inhibitor: DT, drug therapy
serotonin uptake inhibitor: PD, pharmacology
venlafaxine: CM, drug comparison
venlafaxine: DT, drug therapy
venlafaxine: PD, pharmacology
placebo
fluoxetine: CB, drug combination
fluoxetine: CM, drug comparison
fluoxetine: DT, drug therapy
oxcarbazepine: CB, drug combination
oxcarbazepine: DT, drug therapy
clonazepam
lorazepam
topiramate
zonisamide
naltrexone
acamprosate: AE, adverse drug reaction
acamprosate: DT, drug therapy
acamprosate: PD, pharmacology
dihydropyridine
noradrenalin uptake inhibitor: CB, drug combination
modafinil: DT, drug therapy
modafinil: PD, pharmacology
gabapentin
duloxetine: DT, drug therapy
liothyronine
cholinergic receptor stimulating agent
memantine: DT, drug therapy
memantine: PD, pharmacology
folic acid: IT, drug interaction
folic acid: PD, pharmacology
ascorbic acid: PD, pharmacology
vitamin D
calcium
zinc: CB, drug combination
selenium: CB, drug combination
mifepristone
ketoconazole
corticotropin releasing factor antagonist
liothyronine sodium
levothyroxine sodium
pindolol: CB, drug combination
pindolol: DT, drug therapy
pindolol: PD, pharmacology
atomoxetine: AE, adverse drug reaction
atomoxetine: DT, drug therapy

atomoxetine: PD, pharmacology
nutraceutical
chromium picolinate
Rhodiola extract
inositol
choline
n methyl dextro aspartic acid receptor blocking agent
riluzole: DT, drug therapy
riluzole: PD, pharmacology
glycine: AE, adverse drug reaction
glycine: DT, drug therapy
glycine: PD, pharmacology
dextro serine: AE, adverse drug reaction
dextro serine: DT, drug therapy
dextro serine: PD, pharmacology
cycloserine: PD, pharmacology
levothyroxine: DO, drug dose
levothyroxine: PK, pharmacokinetics
buspirone: AE, adverse drug reaction
buspirone: CB, drug combination
buspirone: DT, drug therapy
buspirone: PD, pharmacology
pramipexole: AE, adverse drug reaction
pramipexole: DT, drug therapy
pramipexole: PD, pharmacology
amphetamine: AE, adverse drug reaction
amphetamine: DT, drug therapy
amphetamine: PD, pharmacology
methylphenidate: AE, adverse drug reaction
methylphenidate: DT, drug therapy
methylphenidate: PD, pharmacology
desipramine: AE, adverse drug reaction
desipramine: DT, drug therapy
desipramine: PD, pharmacology
nortriptyline: AE, adverse drug reaction
nortriptyline: DT, drug therapy
nortriptyline: PD, pharmacology
amantadine: AE, adverse drug reaction
amantadine: DT, drug therapy
amantadine: PD, pharmacology

RN (lithium) 7439-93-2; (carbamazepine) 298-46-4, 8047-84-5; (valproic acid) 1069-66-5, 99-66-1; (lamotrigine) 84057-84-1; (molindone) 15622-65-8, 7416-34-4; (clozapine) 5786-21-0; (risperidone) 106266-06-2; (olanzapine) 132539-06-1; (quetiapine) 111974-72-2; (ziprasidone) 118289-78-4, 122883-93-6, 138982-67-9, 199191-69-0; (aripiprazole) 129722-12-9; (imipramine) 113-52-0, 50-49-7; (amfebutamone) 31677-93-7, 34911-55-2; (venlafaxine) 93413-69-5; (fluoxetine) 54910-89-3, 56296-78-7, 59333-67-4; (oxcarbazepine) 28721-07-5; (clonazepam) 1622-61-3; (lorazepam) 846-49-1; (topiramate) 97240-79-4; (zonisamide) 68291-97-4; (naltrexone) 16590-41-3, 16676-29-2; (acamprosate) 77337-73-6; (dihydropyridine) 27790-75-6; (modafinil) 68693-11-8; (gabapentin) 60142-96-3; (duloxetine) 116539-59-4, 136434-34-9; (liothyronine) 6138-47-2, 6893-02-3; (memantine) 19982-08-2, 41100-52-1; (folic acid) 59-30-3, 6484-89-5; (ascorbic acid) 134-03-2, 15421-15-5, 50-81-7; (calcium) 7440-70-2; (zinc) 7440-66-6; (selenium) 7782-49-2; (mifepristone) 84371-65-3; (ketoconazole) 65277-42-1; (liothyronine sodium) 55-06-1; (levothyroxine sodium) 55-03-8; (pindolol) 13523-86-9, 21870-06-4; (atomoxetine) 82248-59-7, 82857-39-4, 82857-40-7, 83015-26-3; (chromium picolinate) 14639-25-9; (inositol) 55608-27-0, 6917-35-7, 87-89-8; (choline) 123-41-1, 13232-47-8, 1927-06-6, 4858-96-2, 62-49-7, 67-48-1; (riluzole) 1744-22-5; (glycine) 56-40-6, 6000-43-7,

6000-44-8; (cycloserine) 339-72-0, 68-39-3, 68-41-7; (levothyroxine) 51-48-9; (buspirone) 33386-08-2, 36505-84-7; (pramipexole) 104632-26-0; (amphetamine) 1200-47-1, 139-10-6, 156-34-3, 2706-50-5, 300-62-9, 51-62-7, 60-13-9, 60-15-1; (methylphenidate) 113-45-1, 298-59-9; (desipramine) 50-47-5, 58-28-6; (nortriptyline) 72-69-5, 894-71-3; (amantadine) 665-66-7, 768-94-5

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ACCESSION NUMBER: 2006037587 EMBASE
TITLE: Parkinson's disease: Aetiology, diagnosis, and management.
AUTHOR: Leung H.; Mok V.
CORPORATE SOURCE: Dr. V. Mok, Department of Medicine and Therapeutics, Chinese University of Hong Kong, Prince of Wales Hospital, Shatin, Hong Kong. b105934@mailserv.cuhk.edu.hk
SOURCE: Hong Kong Medical Journal, (2005) Vol. 11, No. 6, pp. 476-489. .
Refs: 109
ISSN: 1024-2708 E-ISSN: 1024-2708 CODEN: HKMJF3
COUNTRY: Hong Kong
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
008 Neurology and Neurosurgery
036 Health Policy, Economics and Management
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English; Chinese
ENTRY DATE: Entered STN: 24 Feb 2006
Last Updated on STN: 3 Mar 2006

AB Objective. To review the aetiology, diagnosis, and management of Parkinson's disease, with a local perspective. Data sources. Medline from 1966 onwards, and all major neurological journals and movement disorder journals were searched for evidence on the aetiology, diagnosis, and management of Parkinson's disease. Study selection. Key words for the literature search were "Parkinson's disease" and "Chinese" or "Hong Kong". Data extraction. All relevant articles in English were reviewed. Data synthesis. The number of promising genes for familial Parkinson's disease is still expanding rapidly and there has been a wealth of studies on susceptibility genes for Parkinson's disease. Potential treatment choices include the use of agents thought to be neuroprotective, symptomatic treatment with drugs or surgery, and non-pharmacological treatments. Pharmacological treatment using a dopa-sparing strategy and continuous dopaminergic stimulation is now gaining support to address the issue of long-term motor complications. Surgical treatment with deep brain stimulation is safe and effective for refractory cases and has been increasingly utilised locally. Conclusions. Medical therapy remains the mainstay of treatment and newer agents and treatment approaches are emerging, which will hopefully address the issue of neuroprotection and provide symptomatic treatment with fewer motor complications.

CT Medical Descriptors:
*Parkinson disease: DI, diagnosis
*Parkinson disease: DM, disease management
*Parkinson disease: DT, drug therapy
*Parkinson disease: ET, etiology
*Parkinson disease: SU, surgery
data analysis
MEDLINE
scientific literature
motor dysfunction: SI, side effect

medical research
familial disease
neuroprotection
symptom
genetic susceptibility
dopaminergic transmission
genetics
prevalence
neuroimaging
rating scale
dyskinesia: SI, side effect
nausea: SI, side effect
dizziness: SI, side effect
orthostatic hypotension: SI, side effect
hallucination: SI, side effect
hypotension: SI, side effect
urine retention: SI, side effect
xerostomia: SI, side effect
blurred vision: SI, side effect
constipation: SI, side effect
confusion: SI, side effect
insomnia: SI, side effect
nightmare: SI, side effect
skin defect: SI, side effect
brain hemorrhage: SI, side effect
weight gain
  body weight disorder: SI, side effect
dementia: SI, side effect
liver toxicity: SI, side effect
human
clinical trial
review
Drug Descriptors:
alpha tocopherol: PD, pharmacology
selegiline: DT, drug therapy
selegiline: PD, pharmacology
levodopa: AE, adverse drug reaction
levodopa: CT, clinical trial
levodopa: CM, drug comparison
levodopa: DT, drug therapy
rasagiline: CM, drug comparison
rasagiline: DT, drug therapy
dopamine receptor stimulating agent: AE, adverse drug reaction
dopamine receptor stimulating agent: CT, clinical trial
dopamine receptor stimulating agent: DT, drug therapy
dopamine receptor stimulating agent: PD, pharmacology
minocycline: DT, drug therapy
minocycline: PD, pharmacology
riluzole: DT, drug therapy
carbidopa: CT, clinical trial
carbidopa: DT, drug therapy
carbidopa: PK, pharmacokinetics
benserazide: DT, drug therapy
benserazide: PK, pharmacokinetics
bromocriptine: DT, drug therapy
lisuride: DT, drug therapy
lisuride: SC, subcutaneous drug administration
pergolide: DT, drug therapy
cabergoline: DT, drug therapy
  pramipexole: CT, clinical trial

pramipexole: DT, drug therapy
ropinirole: DT, drug therapy
apomorphine: DT, drug therapy
apomorphine: SC, subcutaneous drug administration
piribedil: DT, drug therapy
rotigotine: CT, clinical trial
rotigotine: DT, drug therapy
rotigotine: TD, transdermal drug administration
entacapone: CT, clinical trial
entacapone: DT, drug therapy
entacapone: PK, pharmacokinetics
tolcapone: AE, adverse drug reaction
tolcapone: DT, drug therapy
tolcapone: PK, pharmacokinetics
trihexyphenidyl: DT, drug therapy
amantadine: AE, adverse drug reaction
amantadine: DT, drug therapy
ergot derivative: DT, drug therapy
dopamine receptor
catechol methyltransferase inhibitor: AE, adverse drug reaction
catechol methyltransferase inhibitor: CT, clinical trial
catechol methyltransferase inhibitor: DT, drug therapy
cholinergic receptor blocking agent: AE, adverse drug reaction
cholinergic receptor blocking agent: DT, drug therapy
cholinergic receptor blocking agent: PD, pharmacology
monoamine oxidase inhibitor
adenosine A2 receptor: CT, clinical trial
adenosine A2 receptor: DT, drug therapy
GABAergic receptor affecting agent: CT, clinical trial
unindexed drug
carbidopa plus entacapone plus levodopa
carbidopa plus levodopa
benserazide plus levodopa
bromocriptine mesilate
lisuride maleate
2 (3,5 di tert butyl 4 hydroxyphenyl) 1,1 ethanebisphosphonic acid tetraisopropyl ester
pergolide mesilate

RN (alpha tocopherol) 1406-18-4, 1406-70-8, 52225-20-4, 58-95-7, 59-02-9; (selegiline) 14611-51-9, 14611-52-0, 2079-54-1, 2323-36-6; (levodopa) 59-92-7; (rasagiline) 136236-51-6, 161735-79-1; (minocycline) 10118-90-8, 11006-27-2, 13614-98-7; (riluzole) 1744-22-5; (carbidopa) 28860-95-9; (benserazide) 14919-77-8, 322-35-0; (bromocriptine) 25614-03-3; (lisuride) 18016-80-3; (pergolide) 66104-22-1; (cabergoline) 81409-90-7; (pramipexole) 104632-26-0; (ropinirole) 91374-21-9; (apomorphine) 314-19-2, 58-00-4; (piribedil) 3605-01-4; (rotigotine) 92206-54-7; (entacapone) 116314-67-1; (tolcapone) 134308-13-7; (trihexyphenidyl) 144-11-6, 52-49-3; (amantadine) 665-66-7, 768-94-5; (carbidopa plus levodopa) 57308-51-7; (benserazide plus levodopa) 37270-69-2; (bromocriptine mesilate) 22260-51-1; (lisuride maleate) 19875-60-6; (pergolide mesilate) 66104-23-2

L29 ANSWER 10 OF 27 EMBASE

ACCESSION NUMBER: 2005173422 EMBASE
TITLE: Pharmacological treatment of disabling tremor.
AUTHOR: Schadt C.R.; Duffis E.I.; Charles P.D.
CORPORATE SOURCE: Dr. P.D. Charles, Vanderbilt University Medical Center, The Movement Disorders Clinic, 2100 Pierce Avenue, Nashville, TN 37212-3375, United States. David.Charles@Vanderbilt.edu

SOURCE: Expert Opinion on Pharmacotherapy, (2005) Vol. 6, No. 3, pp. 419-428.
Refs: 86
ISSN: 1465-6566 CODEN: EOPHF7
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 008 Neurology and Neurosurgery
030 Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 5 May 2005
Last Updated on STN: 5 May 2005

AB Tremor is often a disabling primary condition or secondary to another disorder. No universally effective pharmacological agent exists for the treatment of essential tremor, and patients differ greatly in their response to therapies, thus requiring individualised regimens. Deep brain stimulation is the best option for patients with disabling, drug-resistant essential tremor. Resting tremor in Parkinson's disease is usually not the primary disabling feature, and in most cases, levodopa/carbidopa is satisfactory for many years. Young Parkinson's patients with dominant, disabling tremor benefit from anticholinergics in addition to dopaminergic therapies. However, older Parkinson's patients with more disabling tremor may suffer from dose-dependent side effects, and deep brain stimulation should be considered. This article outlines the available pharmacological agents and treatment considerations for various disabling tremors, including essential tremor and Parkinson's disease. .COPYRGT. 2005 Ashley Publications Ltd.

CT Medical Descriptors:
*tremor: DR, drug resistance
*tremor: DT, drug therapy
*tremor: TH, therapy
treatment planning
physical disability
essential tremor: DR, drug resistance
essential tremor: DT, drug therapy
essential tremor: TH, therapy
Parkinson disease: DR, drug resistance
Parkinson disease: DT, drug therapy
Parkinson disease: TH, therapy
drug response
individualization
brain depth stimulation
medical decision making
motor dysfunction: DR, drug resistance
motor dysfunction: DT, drug therapy
motor dysfunction: TH, therapy
drug efficacy
bradykinesia: DT, drug therapy
bradykinesia: TH, therapy
muscle rigidity: DT, drug therapy
muscle rigidity: TH, therapy
dystonia: DT, drug therapy
bradycardia: SI, side effect
syncope: SI, side effect
fatigue: SI, side effect
impotence: SI, side effect
dose response
drug tolerability

nausea: SI, side effect
vomiting: SI, side effect
ataxia: SI, side effect
vertigo: SI, side effect
dizziness: SI, side effect
sedation
somnolence: SI, side effect
mental disease: SI, side effect
disease exacerbation: SI, side effect
substitution therapy
drug dose regimen
confusion: SI, side effect
xerostomia: SI, side effect
visual disorder: SI, side effect
constipation: SI, side effect
cognitive defect: SI, side effect
glaucoma: SI, side effect
urinary tract disease: SI, side effect
insomnia: SI, side effect
valvular heart disease: SI, side effect
drug mechanism
drug half life
edema: SI, side effect
livedo reticularis: SI, side effect
liver dysfunction: SI, side effect
agranulocytosis: SI, side effect
brain disease: DR, drug resistance
brain disease: DT, drug therapy
brain disease: TH, therapy
alcohol withdrawal: DT, drug therapy
paresthesia: SI, side effect
libido disorder: SI, side effect
nervousness
dyspnea: SI, side effect
maximum tolerated dose
weight reduction
  body weight disorder: SI, side effect
human
clinical trial
review

CT Drug Descriptors:
carbidopa plus levodopa: CT, clinical trial
carbidopa plus levodopa: CM, drug comparison
carbidopa plus levodopa: DO, drug dose
carbidopa plus levodopa: DT, drug therapy
cholinergic receptor blocking agent: AE, adverse drug reaction
cholinergic receptor blocking agent: CB, drug combination
cholinergic receptor blocking agent: CM, drug comparison
cholinergic receptor blocking agent: DT, drug therapy
dopamine receptor stimulating agent: AE, adverse drug reaction
dopamine receptor stimulating agent: CT, clinical trial
dopamine receptor stimulating agent: CB, drug combination
dopamine receptor stimulating agent: DT, drug therapy
propranolol: AE, adverse drug reaction
propranolol: CT, clinical trial
propranolol: CM, drug comparison
propranolol: DO, drug dose
propranolol: DT, drug therapy
primidone: AE, adverse drug reaction
primidone: CT, clinical trial

primidone: CM, drug comparison
primidone: DO, drug dose
primidone: DT, drug therapy
botulinum toxin A: CT, clinical trial
botulinum toxin A: DO, drug dose
botulinum toxin A: DT, drug therapy
naxagolide: CT, clinical trial
naxagolide: CM, drug comparison
naxagolide: DO, drug dose
naxagolide: DT, drug therapy
trihexyphenidyl: CT, clinical trial
trihexyphenidyl: CB, drug combination
trihexyphenidyl: CM, drug comparison
trihexyphenidyl: DO, drug dose
trihexyphenidyl: DT, drug therapy
levodopa: AE, adverse drug reaction
levodopa: CT, clinical trial
levodopa: CB, drug combination
levodopa: CM, drug comparison
levodopa: DT, drug therapy
biperiden: CT, clinical trial
biperiden: CM, drug comparison
biperiden: DO, drug dose
biperiden: DT, drug therapy
apomorphine: CT, clinical trial
apomorphine: CM, drug comparison
apomorphine: DO, drug dose
apomorphine: DT, drug therapy
procyclidine: AE, adverse drug reaction
procyclidine: CM, drug comparison
procyclidine: DT, drug therapy
pramipexole: AE, adverse drug reaction
pramipexole: CT, clinical trial
pramipexole: DO, drug dose
pramipexole: DT, drug therapy
pergolide: AE, adverse drug reaction
pergolide: CT, clinical trial
pergolide: CM, drug comparison
pergolide: DT, drug therapy
bromocriptine: AE, adverse drug reaction
bromocriptine: CT, clinical trial
bromocriptine: CM, drug comparison
bromocriptine: DT, drug therapy
lisuride: CT, clinical trial
lisuride: CM, drug comparison
lisuride: DT, drug therapy
selegiline: CT, clinical trial
selegiline: CB, drug combination
selegiline: CM, drug comparison
selegiline: DO, drug dose
selegiline: DT, drug therapy
amantadine: AE, adverse drug reaction
amantadine: CT, clinical trial
amantadine: CM, drug comparison
amantadine: DT, drug therapy
amantadine: PK, pharmacokinetics
amantadine: PD, pharmacology
tolcapone: AE, adverse drug reaction
tolcapone: CM, drug comparison
tolcapone: DO, drug dose

tolcapone: DT, drug therapy
alcohol
isoniazid: CT, clinical trial
isoniazid: CB, drug combination
isoniazid: DO, drug dose
isoniazid: DT, drug therapy
pyridoxine: CT, clinical trial
pyridoxine: CB, drug combination
pyridoxine: DO, drug dose
pyridoxine: DT, drug therapy
carbamazepine: CT, clinical trial
carbamazepine: DO, drug dose
carbamazepine: DT, drug therapy
clonazepam: AE, adverse drug reaction
clonazepam: CT, clinical trial
clonazepam: DO, drug dose
clonazepam: DT, drug therapy
gabapentin: AE, adverse drug reaction
gabapentin: CT, clinical trial
gabapentin: CM, drug comparison
gabapentin: DO, drug dose
gabapentin: DT, drug therapy
benzatropine: DT, drug therapy
alprazolam: AE, adverse drug reaction
alprazolam: CT, clinical trial
alprazolam: CM, drug comparison
alprazolam: DO, drug dose
alprazolam: DT, drug therapy
alprazolam: PK, pharmacokinetics
topiramate: AE, adverse drug reaction
topiramate: CT, clinical trial
topiramate: DO, drug dose
topiramate: DT, drug therapy
clozapine: AE, adverse drug reaction
clozapine: CT, clinical trial
clozapine: DO, drug dose
clozapine: DT, drug therapy
unindexed drug

RN (carbidopa plus levodopa) 57308-51-7; (propranolol) 13013-17-7, 318-98-9, 3506-09-0, 4199-09-1, 525-66-6; (primidone) 125-33-7; (botulinum toxin A) 93384-43-1; (naxagolide) 88058-88-2; (trihexyphenidyl) 144-11-6, 52-49-3; (levodopa) 59-92-7; (biperiden) 1235-82-1, 514-65-8; (apomorphine) 314-19-2, 58-00-4; (procyclidine) 1508-76-5, 77-37-2; (pramipexole) 104632-26-0; (pergolide) 66104-22-1; (bromocriptine) 25614-03-3; (lisuride) 18016-80-3; (selegiline) 14611-51-9, 14611-52-0, 2079-54-1, 2323-36-6; (amantadine) 665-66-7, 768-94-5; (tolcapone) 134308-13-7; (alcohol) 64-17-5; (isoniazid) 54-85-3, 62229-51-0, 65979-32-0; (pyridoxine) 12001-77-3, 58-56-0, 65-23-6, 8059-24-3; (carbamazepine) 298-46-4, 8047-84-5; (clonazepam) 1622-61-3; (gabapentin) 60142-96-3; (benzatropine) 86-13-5; (alprazolam) 28981-97-7; (topiramate) 97240-79-4; (clozapine) 5786-21-0

L29 ANSWER 11 OF 27 EMBASE

ACCESSION NUMBER: 2005418892 EMBASE
TITLE: Effective maintenance treatment - Breaking the cycle of bipolar disorder.
AUTHOR: Goodwin G.; Vieta E.
CORPORATE SOURCE: G. Goodwin, Department of Psychiatry, Warneford Hospital, University of Oxford, Oxford, United Kingdom.

Prepared by: Mary Hale @2-2507 Rem Bldg 1D86

guy.goodwin@psych.ox.ac.uk
SOURCE: European Psychiatry, (2005) Vol. 20, No. 5-6, pp. 365-371.
Refs: 25
ISSN: 0924-9338 CODEN: EUPSED
PUBLISHER IDENT.: S 0924-9338(05)00120-3
COUNTRY: France
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 032 Psychiatry
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 13 Oct 2005
Last Updated on STN: 13 Oct 2005

AB Clinical guidelines for treatment and research of bipolar disorder greatly benefit from the synthesis of data from individual studies. The British Association for Psychopharmacology bases its guidelines on evidence from opinions (level D) to systematic reviews of primary trial data (level A). The report details conclusions of its 1-day consensus meeting to develop guidelines covering diagnosis, clinical management, pharmacotherapy for acute episodes, relapse prevention and treatment discontinuation. Monotherapy for long-term management is preferred, having reduced side-effects and drug interactions and improved compliance. Combination therapy is often preferred for acute episodes, using antipsychotics for mania or antidepressants for depression. Increased efficacy may be attributed to multiple mechanisms of action and potentially lower doses. In clinical practice, maintenance monotherapy has limited success for chronic episodes and polypharmacy is frequently used, though the best combination remains unclear. A new collaborative approach based on simple clinical trials is required to change current medical practice.

CT Medical Descriptors:
*bipolar disorder: DT, drug therapy
*maintenance therapy
*psychopharmacotherapy
practice guideline
recurrent disease
monotherapy
long term care
patient compliance
mania: DT, drug therapy
depression: DT, drug therapy
drug efficacy
clinical practice
polypharmacy
medical practice
prophylaxis
weight gain
side effect: SI, side effect
obesity: SI, side effect
diabetes mellitus: SI, side effect
extrapyramidal syndrome: SI, side effect
weight reduction
sexual dysfunction: SI, side effect
convalescence
illness behavior
psychological aspect
physician attitude
disease duration

age distribution
patient care
patient education
self medication
human
clinical trial
systematic review
article
priority journal
Drug Descriptors:
neuroleptic agent: CT, clinical trial
neuroleptic agent: CM, drug comparison
neuroleptic agent: DT, drug therapy
neuroleptic agent: PD, pharmacology
antidepressant agent: CM, drug comparison
antidepressant agent: DT, drug therapy
lithium: AE, adverse drug reaction
lithium: CT, clinical trial
lithium: CB, drug combination
lithium: CM, drug comparison
lithium: DT, drug therapy
carbamazepine: CB, drug combination
carbamazepine: IT, drug interaction
carbamazepine: DT, drug therapy
valproic acid: CT, clinical trial
valproic acid: CB, drug combination
valproic acid: DO, drug dose
valproic acid: IT, drug interaction
valproic acid: DT, drug therapy
olanzapine: AE, adverse drug reaction
olanzapine: CT, clinical trial
olanzapine: CB, drug combination
olanzapine: CM, drug comparison
olanzapine: DT, drug therapy
aripiprazole: CB, drug combination
aripiprazole: DT, drug therapy
lamotrigine: AE, adverse drug reaction
lamotrigine: CB, drug combination
lamotrigine: CR, drug concentration
lamotrigine: DO, drug dose
lamotrigine: IT, drug interaction
lamotrigine: DT, drug therapy
haloperidol: CT, clinical trial
haloperidol: CB, drug combination
haloperidol: DT, drug therapy
perazine: CT, clinical trial
perazine: CB, drug combination
perazine: DT, drug therapy
risperidone: CT, clinical trial
risperidone: CB, drug combination
risperidone: DT, drug therapy
quetiapine: CT, clinical trial
quetiapine: CB, drug combination
quetiapine: DT, drug therapy
ziprasidone: CB, drug combination
ziprasidone: DT, drug therapy
pramipexole: CT, clinical trial
pramipexole: CB, drug combination
pramipexole: DT, drug therapy
mood stabilizer: CT, clinical trial

mood stabilizer: CB, drug combination
mood stabilizer: DT, drug therapy
topiramate: AE, adverse drug reaction
topiramate: CB, drug combination
topiramate: DT, drug therapy
anticonvulsive agent: CT, clinical trial
anticonvulsive agent: CB, drug combination
anticonvulsive agent: CM, drug comparison
anticonvulsive agent: IT, drug interaction
anticonvulsive agent: PD, pharmacology
RN (lithium) 7439-93-2; (carbamazepine) 298-46-4, 8047-84-5; (valproic acid) 1069-66-5, 99-66-1; (olanzapine) 132539-06-1; (aripiprazole) 129722-12-9; (lamotrigine) 84057-84-1; (haloperidol) 52-86-8; (perazine) 84-97-9; (risperidone) 106266-06-2; (quetiapine) 111974-72-2; (ziprasidone) 118289-78-4, 122883-93-6, 138982-67-9, 199191-69-0; (pramipexole) 104632-26-0; (topiramate) 97240-79-4

L29 ANSWER 12 OF 27 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2005:487997 BIOSIS
DOCUMENT NUMBER: PREV200510290259
TITLE: Management of augmentatiion in patients with restless legs syndrome.
AUTHOR(S): Trenkwalder, C. [Reprint Author]; Canelo, M.
CORPORATE SOURCE: Paracelsul Elena Klin, Ctr Parkinsonism and Movement Disorders, Kassel, Germany
SOURCE: Sleep (Rochester), (2005) Vol. 28, No. Suppl. S, pp. A278. Meeting Info.: 19th Annual Meeting of the Associated-Professional-Sleep-Societies. Denver, CO, USA. June 18 -23, 2005. Associated Profess Sleep Soc. CODEN: SLEED6. ISSN: 0161-8105.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 16 Nov 2005
Last Updated on STN: 16 Nov 2005
IT Major Concepts
Pharmacology; Neurology (Human Medicine, Medical Sciences)
IT Diseases
diabetes: endocrine disease/pancreas, metabolic disease
Diabetes Mellitus (MeSH)
IT Diseases
polyneuropathy: nervous system disease
Polyneuropathies (MeSH)
IT Diseases
iron deficiency: nutritional disease
IT Diseases
restless legs syndrome: nervous system disease, drug therapy, symptom, diagnosis
IT Chemicals & Biochemicals
pramipexole: antiparkinsonian-drug, dopamine receptor agonist-drug; iron: vitamin-drug, intravenous administration; pergolide: neuroprotectant-drug, dopamine receptor agonist-drug, antiparkinsonian-drug; levodopa: antiparkinsonian-drug, dopamine receptor agonist-drug; tilidine: sedative/hypnotic-drug, opioid; tramadol: sedative/hypnotic-drug, opioid; opioid benzodiazepine: sedative/hypnotic-drug, opioid
RN 104632-26-0 (pramipexole)
7439-89-6 (iron)

Prepared by: Mary Hale @2-2507 Rem Bldg 1D86

66104-22-1 (pergolide)
59-92-7 (levodopa)
51931-66-9 (tilidine)
27203-92-5 (tramadol)

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ACCESSION NUMBER: 2005132066 EMBASE
TITLE: The primary parasomnias: A review for neurologists.
AUTHOR: Giglio P.; Undevia N.; Spire J.-P.
CORPORATE SOURCE: Dr. J.-P. Spire, University of Chicago Hospitals, Department of Neurology, MC 2040, 5841 S. Maryland Avenue, Chicago, IL 60637, United States. jpspire@neurology.bsd.uchicago.edu
SOURCE: Neurologist, (2005) Vol. 11, No. 2, pp. 90-97. .
Refs: 59
ISSN: 1074-7931 CODEN: NROLFW
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 007 Pediatrics and Pediatric Surgery
008 Neurology and Neurosurgery
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 7 Apr 2005
Last Updated on STN: 7 Apr 2005

AB Background: Primary parasomnias are undesirable motor or verbal phenomena which occur during sleep and result in abnormal arousals. They occur out of all sleep stages or during transitions between sleep and awake. Secondary parasomnias are sleep disturbances that are caused by disorders of other organ systems. This review addresses only primary parasomnias. Arousal disorders and the parasomnias associated with REM sleep are the primary parasomnias most likely to be seen in a neurology practice. Sleep-wake transition disorders are also discussed with nocturnal leg cramps, probably the most common in this group. Review Summary: The salient clinical features of the primary parasomnias are discussed. Emphasis is placed on the differential diagnosis of the different conditions and the best management strategies. Parasomnias encountered in infancy, such as infant sleep apnea, are not discussed in this review. Conclusions: Parasomnias are common disturbances of sleep that may significantly affect the patient's quality of life and that of the bed partner. Most parasomnias can be diagnosed with careful history taking and polysomnography, and management is usually safe and effective. Copyright .COPYRGT. 2005 by Lippincott Williams & Wilkins.

CT Medical Descriptors:
*parasomnia: DI, diagnosis
*parasomnia: DT, drug therapy
confusion
arousal
differential diagnosis
REM sleep
sleep terrors
facial expression
seizure
sleep disordered breathing
restless legs syndrome
sleep walking: DT, drug therapy
heredity
family history

sleep deprivation
social problem
somnolence: SI, side effect
rhythmic movement disorder: DT, drug therapy
sleep starts
sleep talking
leg cramp
gastrocnemius muscle
massage
leg movement
nightmare
relaxation training
behavior therapy
penis erection
sleep related painful erection: DT, drug therapy
sinus arrest: TH, therapy
artificial heart pacemaker
behavior disorder
bruxism: DT, drug therapy
nocturnal enuresis: DT, drug therapy
snoring: SU, surgery
risk factor
positive end expiratory pressure
obesity
uvulopalatopharyngoplasty
hemolytic uremic syndrome: SI, side effect
thrombocytopenia: SI, side effect
human
review
priority journal
Drug Descriptors:
benzodiazepine: DT, drug therapy
diazepam: AE, adverse drug reaction
diazepam: DT, drug therapy
tricyclic antidepressant agent: DT, drug therapy
imipramine: DT, drug therapy
clonazepam: AE, adverse drug reaction
clonazepam: DT, drug therapy
vitamin B group: DT, drug therapy
calcium channel blocking agent: DT, drug therapy
quinine sulfate: AE, adverse drug reaction
quinine sulfate: DT, drug therapy
potassium: DT, drug therapy
magnesium citrate: DT, drug therapy
serotonin uptake inhibitor: DT, drug therapy
propranolol: DT, drug therapy
paroxetine: DT, drug therapy
anxiolytic agent: DT, drug therapy
beta adrenergic receptor blocking agent: DT, drug therapy
botulinum toxin: DT, drug therapy
neuroleptic agent
desmopressin: DT, drug therapy
carbidopa plus levodopa
dopamine receptor stimulating agent
pramipexole
RN (benzodiazepine) 12794-10-4; (diazepam) 439-14-5; (imipramine) 113-52-0, 50-49-7; (clonazepam) 1622-61-3; (vitamin B group) 12001-76-2; (quinine sulfate) 804-63-7; (potassium) 7440-09-7; (magnesium citrate) 144-23-0, 3344-18-1, 7779-25-1; (propranolol) 13013-17-7, 318-98-9, 3506-09-0, 4199-09-1, 525-66-6; (paroxetine) 61869-08-7; (desmopressin) 16679-58-6;

(carbidopa plus levodopa) 57308-51-7; (pramipexole) 104632-26-0

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ACCESSION NUMBER: 2006004322 EMBASE
TITLE: Newer treatment studies for bipolar depression.
AUTHOR: Gao K.; Calabrese J.R.
CORPORATE SOURCE: Dr. K. Gao, 11400 Euclid Avenue, Cleveland, OH 44106, United States. keming.gao@uhhs.com
SOURCE: Bipolar Disorders, Supplement, (2005) Vol. 7, No. 5, pp. 13-23. .
Refs: 40
ISSN: 1399-2406 CODEN: BDSICE
COUNTRY: Denmark
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 032 Psychiatry
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 12 Jan 2006
Last Updated on STN: 12 Jan 2006

AB Objective: Depressive symptoms of bipolar disorder have more negative impact on a patient's life than manic symptoms. This review focused on the emerging efficacy data for treatments in bipolar depression. Methods: English-language literature cited in Medline was searched with terms bipolar depression, clinical trial, and trial. Randomized, placebo-controlled trials of newer studies with older agents and all studies with newer or novel agents were prioritized. Open-label studies of novel agents presented at major scientific meetings were also included. Results: Olanzapine, olanzapine-fluoxetine combination (OFC), and quetiapine were superior to placebo in the acute treatment of bipolar depression. Lamotrigine only significantly reduced core symptoms of depression compared with placebo. Pramipexole, a dopamine D2/D3 receptor agonist and omega-3 fatty acids, a polyunsaturated fatty acid, augmentation to mood stabilizer (MS) had superiority to placebo in reducing depressive symptoms. Topiramate augmentation of an MS was equally as effective as Bupropion-SR. Patients treated with an MS responded well to the addition of agomelatine, a melatonin receptor agonist with 5-HT2C antagonist properties. However, inositol and repetitive transcranial magnetic stimulation did not separate from placebo. Lamotrigine and olanzapine, and to a lesser extent, divalproex, are superior to placebo in preventing depressive relapses. All agents were relatively well tolerated. Conclusions: Olanzapine, OFC, and quetiapine are effective in the acute treatment of bipolar depression. Compared with lithium and divalproex, lamotrigine is more effective in preventing bipolar depression. Larger controlled studies of the other agents in the acute and maintenance treatment of bipolar depression are warranted. .COPYRGT. Blackwell Munksgaard, 2005.

AB Objective: Depressive symptoms of bipolar disorder have more negative impact on a patient's life than manic symptoms. This review focused on the emerging efficacy data for treatments in bipolar depression. Methods: English-language literature cited in Medline was searched with terms bipolar depression, clinical trial, and trial. Randomized, placebo-controlled trials of newer studies with older agents and all studies with newer or novel agents were prioritized. Open-label studies of novel agents presented at major scientific meetings were also included. Results: Olanzapine, olanzapine-fluoxetine combination (OFC), and quetiapine were superior to placebo in the acute treatment of bipolar depression. Lamotrigine only significantly reduced core symptoms of

depression compared with placebo. Pramipexole, a dopamine D2/D3 receptor agonist and omega-3 fatty acids, a polyunsaturated fatty acid, augmentation to mood stabilizer (MS) had superiority to placebo in reducing depressive symptoms. Topiramate augmentation of an MS was equally as effective as Bupropion-SR. Patients treated with an MS responded well to the addition of agomelatine, a melatonin receptor agonist with 5-HT2C antagonist properties. However, inositol and repetitive transcranial magnetic stimulation did not separate from placebo. Lamotrigine and olanzapine, and to a lesser extent, divalproex, are superior to placebo in preventing depressive relapses. All agents were relatively well tolerated. Conclusions: Olanzapine, OFC, and quetiapine are effective in the acute treatment of bipolar depression. Compared with lithium and divalproex, lamotrigine is more effective in preventing bipolar depression. Larger controlled studies of the other agents in the acute and maintenance treatment of bipolar depression are warranted. .COPYRGT. Blackwell Munksgaard, 2005.

CT Medical Descriptors:
*bipolar depression: DT, drug therapy
*bipolar depression: TH, therapy
bipolar disorder
bipolar mania
drug efficacy
MEDLINE
open study
drug potentiation
add on therapy
transcranial magnetic stimulation
relapse
drug tolerability
maintenance therapy
headache: SI, side effect
rash: SI, side effect
anxiety disorder: SI, side effect
appetite disorder: SI, side effect
visual impairment: SI, side effect
xerostomia: SI, side effect
memory disorder: SI, side effect
nausea: SI, side effect
nervousness
emotional disorder: SI, side effect
paresthesia: SI, side effect
sweating
sweat gland disease: SI, side effect
tremor: SI, side effect
language disability: SI, side effect
sleep disorder: SI, side effect
weight reduction
  body weight disorder: SI, side effect
somnolence: SI, side effect
weight gain
increased appetite: SI, side effect
asthenia: SI, side effect
dizziness: SI, side effect
constipation: SI, side effect
mania: SI, side effect
insomnia: SI, side effect
vomiting: SI, side effect
restlessness: SI, side effect
gastrointestinal symptom: SI, side effect
lassitude: SI, side effect

hypomania
disease exacerbation: SI, side effect
diarrhea: DT, drug therapy
diarrhea: SI, side effect
Stevens Johnson syndrome: SI, side effect
fatigue: SI, side effect
toxic epidermal necrolysis: SI, side effect
human
clinical trial
review
priority journal
CT Drug Descriptors:
*olanzapine: AE, adverse drug reaction
*olanzapine: CT, clinical trial
*olanzapine: CB, drug combination
*olanzapine: CM, drug comparison
*olanzapine: DT, drug therapy
*fluoxetine plus olanzapine: AE, adverse drug reaction
*fluoxetine plus olanzapine: CT, clinical trial
*fluoxetine plus olanzapine: CM, drug comparison
*fluoxetine plus olanzapine: DT, drug therapy
*quetiapine: AE, adverse drug reaction
*quetiapine: CT, clinical trial
*quetiapine: DO, drug dose
*quetiapine: DT, drug therapy
*lamotrigine: AE, adverse drug reaction
*lamotrigine: CT, clinical trial
*lamotrigine: CM, drug comparison
*lamotrigine: DT, drug therapy
*pramipexole: AE, adverse drug reaction
*pramipexole: CT, clinical trial
*pramipexole: CB, drug combination
*pramipexole: DT, drug therapy
*pramipexole: PD, pharmacology
placebo
omega 3 fatty acid: AE, adverse drug reaction
omega 3 fatty acid: CT, clinical trial
omega 3 fatty acid: CB, drug combination
omega 3 fatty acid: DT, drug therapy
mood stabilizer: CT, clinical trial
mood stabilizer: CB, drug combination
mood stabilizer: CM, drug comparison
mood stabilizer: IT, drug interaction
mood stabilizer: DT, drug therapy
topiramate: AE, adverse drug reaction
topiramate: CT, clinical trial
topiramate: CB, drug combination
topiramate: CM, drug comparison
topiramate: DO, drug dose
topiramate: IT, drug interaction
topiramate: DT, drug therapy
amfebutamone: AE, adverse drug reaction
amfebutamone: CT, clinical trial
amfebutamone: CB, drug combination
amfebutamone: CM, drug comparison
amfebutamone: DO, drug dose
amfebutamone: DT, drug therapy
agomelatine: CB, drug combination
agomelatine: DT, drug therapy
agomelatine: PD, pharmacology

Prepared by: Mary Hale @2-2507 Rem Bldg 1D86

inositol: CT, clinical trial
inositol: CM, drug comparison
inositol: DT, drug therapy
valproate semisodium: CT, clinical trial
valproate semisodium: CB, drug combination
valproate semisodium: CM, drug comparison
valproate semisodium: DT, drug therapy
lithium: CT, clinical trial
lithium: CB, drug combination
lithium: CM, drug comparison
lithium: DT, drug therapy
carbamazepine: CM, drug comparison
carbamazepine: DT, drug therapy
antidepressant agent: CB, drug combination
antidepressant agent: CM, drug comparison
antidepressant agent: DT, drug therapy
glucose
cholinergic receptor blocking agent: DT, drug therapy
valproic acid: CT, clinical trial
valproic acid: DT, drug therapy
paroxetine: DT, drug therapy
sertraline: DT, drug therapy
serotonin uptake inhibitor

RN (olanzapine) 132539-06-1; (quetiapine) 111974-72-2; (lamotrigine) 84057-84-1; (pramipexole) 104632-26-0; (topiramate) 97240-79-4; (amfebutamone) 31677-93-7, 34911-55-2; (agomelatine) 138112-76-2; (inositol) 55608-27-0, 6917-35-7, 87-89-8; (valproate semisodium) 76584-70-8; (lithium) 7439-93-2; (carbamazepine) 298-46-4, 8047-84-5; (glucose) 50-99-7, 84778-64-3; (valproic acid) 1069-66-5, 99-66-1; (paroxetine) 61869-08-7; (sertraline) 79617-96-2

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ACCESSION NUMBER: 2005357697 EMBASE
TITLE: Fibromyalgia and chronic fatigue syndrome.
AUTHOR: Fan P.T.
CORPORATE SOURCE: Dr. P.T. Fan, Division of Rheumatology, David Geffen School of Medicine at UCLA, Los Angeles, CA, United States. cw4@mindspring.com
SOURCE: APLAR Journal of Rheumatology, (2004) Vol. 7, No. 3, pp. 219-231.
Refs: 93
ISSN: 0219-0494 CODEN: AJRPBQ
COUNTRY: Australia
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
006 Internal Medicine
031 Arthritis and Rheumatism
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 9 Sep 2005
Last Updated on STN: 9 Sep 2005

AB Fibromyalgia (FM) is a common disorder that affects 3% of the general population. Chronic fatigue syndrome (CFS) is less common, with about 1% of adults in the US meeting the current CDC criteria for case definition. Both conditions are controversial because they do not fit either a strictly physical or psychological concept of disease. Their symptoms overlap and they share many clinical features with irritable bowel syndrome, atypical migraine and muscle tension headaches, multiple

chemical sensitivities, interstitial cystitis and multiple allergies syndrome, all poorly understood entities. This review provides an historical perspective on both disorders and presents the clinical features and differential diagnoses. The relative success of current classification criteria is discussed. Pathogenic mechanisms are presented, including the relationship of FM and CFS to depression and other psychiatric diseases and the possibility that they may be 'diseases constructs' called 'memes' that are invented by physicians. A cognitive-behavioral approach to treatment is presented that emphasizes the roles of education, stress reduction, improvement of sleep, exercise and the effective use of analgesics, antidepressants and other psychoactive medications.

CT Medical Descriptors:
*fibromyalgia: DI, diagnosis
*fibromyalgia: DT, drug therapy
*fibromyalgia: EP, epidemiology
*fibromyalgia: ET, etiology
*chronic fatigue syndrome: DI, diagnosis
*chronic fatigue syndrome: DT, drug therapy
*chronic fatigue syndrome: EP, epidemiology
*chronic fatigue syndrome: ET, etiology
clinical feature
differential diagnosis
pathogenesis
depression: DT, drug therapy
mental disease: DT, drug therapy
physician
stress
sleep
exercise
pain
exhaustion
sleep deprivation
fatigue
lassitude
anxiety
paresthesia
dizziness
vertigo
muscle cramp
bloating
muscle spasm
pelvis pain syndrome
bladder disease
cognitive defect
memory disorder
heart palpitation
dyspnea
vulvodynia
body weight
night sweat
muscle weakness
sore throat
cervical lymph node
myalgia
arthralgia
malaise
upper respiratory tract infection
patient education

injection
muscle stretching
prognosis
history
disease classification
drug efficacy
drug use
human
review
priority journal
Drug Descriptors:
analgesic agent: DT, drug therapy
psychotropic agent: DT, drug therapy
lidocaine: DT, drug therapy
corticosteroid: DT, drug therapy
sodium chloride: DT, drug therapy
botulinum toxin: DT, drug therapy
cyclobenzaprine: DT, drug therapy
doxepin: DO, drug dose
doxepin: DT, drug therapy
nonsteroid antiinflammatory agent: DT, drug therapy
cyclooxygenase 2 inhibitor: DT, drug therapy
prednisone: DT, drug therapy
tramadol: DT, drug therapy
paracetamol: DT, drug therapy
antidepressant agent: CB, drug combination
antidepressant agent: DT, drug therapy
fluoxetine: CB, drug combination
fluoxetine: DT, drug therapy
amitriptyline: CB, drug combination
amitriptyline: DT, drug therapy
zolpidem: DT, drug therapy
temazepam: DT, drug therapy
melatonin: DT, drug therapy
benzodiazepine: DT, drug therapy
clonazepam: DT, drug therapy
carbidopa plus levodopa: DT, drug therapy
pramipexole: DT, drug therapy
modafinil: DT, drug therapy
pemoline: DT, drug therapy
methylphenidate: DT, drug therapy
fludrocortisone: DT, drug therapy
midodrine: DT, drug therapy
nicotinamide adenine dinucleotide: DT, drug therapy
unindexed drug
RN (lidocaine) 137-58-6, 24847-67-4, 56934-02-2, 73-78-9; (sodium chloride) 7647-14-5; (cyclobenzaprine) 303-53-7, 6202-23-9; (doxepin) 1229-29-4, 1668-19-5; (prednisone) 53-03-2; (tramadol) 27203-92-5, 36282-47-0; (paracetamol) 103-90-2; (fluoxetine) 54910-89-3, 56296-78-7, 59333-67-4; (amitriptyline) 50-48-6, 549-18-8; (zolpidem) 82626-48-0; (temazepam) 846-50-4; (melatonin) 73-31-4; (benzodiazepine) 12794-10-4; (clonazepam) 1622-61-3; (carbidopa plus levodopa) 57308-51-7; (pramipexole) 104632-26-0; (modafinil) 68693-11-8; (pemoline) 2152-34-3; (methylphenidate) 113-45-1, 298-59-9; (fludrocortisone) 127-31-1; (midodrine) 3092-17-9, 42794-76-3; (nicotinamide adenine dinucleotide) 53-84-9

L29 ANSWER 16 OF 27 EMBASE
ACCESSION NUMBER: 2004299492 EMBASE

Prepared by: Mary Hale @2-2507 Rem Bldg 1D86

TITLE: Restless legs syndrome.
AUTHOR: Lesage S.; Earley C.J.
CORPORATE SOURCE: Dr. S. Lesage, Department of Neurology, J. Hopkins Bayview Medical Center, J. Hopkins Ctr. Restless Legs Synd., 5501 Hopkins Bayview Circle, Baltimore, MD 21224, United States. slesage@jhmi.edu
SOURCE: Current Treatment Options in Neurology, (2004) Vol. 6, No. 3, pp. 209-219. .
Refs: 47
ISSN: 1092-8480 CODEN: CTONBT
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 008 Neurology and Neurosurgery
030 Pharmacology
036 Health Policy, Economics and Management
037 Drug Literature Index
038 Adverse Reactions Titles
039 Pharmacy
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 29 Jul 2004
Last Updated on STN: 29 Jul 2004
AB In the past 10 years, restless legs syndrome (RLS) has gained recognition as a common sleep disorder. There are several therapeutic options in treating patients with RLS. RLS causes significant sleep distrubance and negatively impacts on patient quality of life. Pharmacologic treatment can result in improved sleep and quality of life issues. RLS patients should be evaluated for iron deficiency anemia; iron replacement in defecient patients may lead to a resolution of symptoms or may reduce the severity of their symptoms. For patients with daily symptoms, the initial therapy is dopamine agonists. Low doses given in the evening or 2 hours before bed provide adequate relief of symptoms for many RLS patients. Augmentation can be seen with all dopamine agents, but is most prevalent with levodopa. Levodopa therapy is best used for milder intermittent symptoms or in aggravating situations, such as long car rides. Opiates and antiepileptics remain a beneficial therapy for RLS and are useful in patients who experience pain as part of their RLS. Newer anticonvulsants may provide additional treatment options, but they have yet to undergo clinical trials. Intravenous iron also may provide relief of RLS symptoms; however, dosing and safety issues have not been fully evaluated in a RLS population.
CT Medical Descriptors:
*restless legs syndrome: DI, diagnosis
*restless legs syndrome: DM, disease management
*restless legs syndrome: DT, drug therapy
*restless legs syndrome: ET, etiology
sleep disorder
patient care
quality of life
iron deficiency anemia: CO, complication
iron deficiency anemia: DT, drug therapy
disease severity
low drug dose
drug dose regimen
disease exacerbation: DT, drug therapy
drug blood level
polysomnography
dose response
constipation: SI, side effect
diarrhea: SI, side effect

abdominal discomfort: SI, side effect
nausea: SI, side effect
vomiting: SI, side effect
drug absorption
drug cost
drug contraindication
orthostatic hypertension: SI, side effect
gastrointestinal symptom: SI, side effect
somnolence: SI, side effect
dizziness: SI, side effect
mental disease: SI, side effect
headache: SI, side effect
muscle cramp: SI, side effect
protein binding
abdominal pain: SI, side effect
fluid retention
rhinitis: SI, side effect
dyspnea: SI, side effect
drug eruption: SI, side effect
insomnia: SI, side effect
drug half life
drug excretion
malaise: SI, side effect
xerostomia: SI, side effect
peripheral edema: SI, side effect
body weight disorder: SI, side effect
diplopia: SI, side effect
ataxia: SI, side effect
nervousness
tremor: SI, side effect
dysarthria: SI, side effect
respiration depression: SI, side effect
central nervous system depression
confusion: SI, side effect
excitability
irritability
euphoria
dysphoria: SI, side effect
urine retention: SI, side effect
pruritus: SI, side effect
visual disorder: SI, side effect
cost effectiveness analysis
human
clinical trial
review

CT Drug Descriptors:
iron: AE, adverse drug reaction
iron: CT, clinical trial
iron: CB, drug combination
iron: CR, drug concentration
iron: DO, drug dose
iron: IT, drug interaction
iron: DT, drug therapy
iron: PK, pharmacokinetics
iron: IV, intravenous drug administration
dopamine receptor stimulating agent: AE, adverse drug reaction
dopamine receptor stimulating agent: DO, drug dose
dopamine receptor stimulating agent: IT, drug interaction
dopamine receptor stimulating agent: DT, drug therapy
dopamine receptor stimulating agent: PE, pharmacoeconomics

levodopa: AE, adverse drug reaction
levodopa: CB, drug combination
levodopa: DO, drug dose
levodopa: IT, drug interaction
levodopa: DT, drug therapy
levodopa: PE, pharmacoeconomics
levodopa: PR, pharmaceutics
opiate: AE, adverse drug reaction
opiate: CT, clinical trial
opiate: DO, drug dose
opiate: IT, drug interaction
opiate: DT, drug therapy
opiate: PE, pharmacoeconomics
anticonvulsive agent: DT, drug therapy
benzodiazepine derivative: DT, drug therapy
gabapentin: AE, adverse drug reaction
gabapentin: CT, clinical trial
gabapentin: DO, drug dose
gabapentin: IT, drug interaction
gabapentin: DT, drug therapy
gabapentin: PE, pharmacoeconomics
gabapentin: PK, pharmacokinetics
carbamazepine: DT, drug therapy
clonazepam: AE, adverse drug reaction
clonazepam: DO, drug dose
clonazepam: IT, drug interaction
clonazepam: DT, drug therapy
clonazepam: PE, pharmacoeconomics
clonazepam: PD, pharmacology
caffeine
alcohol
ferritin: EC, endogenous compound
tetracycline: IT, drug interaction
tetracycline: PK, pharmacokinetics
tetracycline: PO, oral drug administration
ascorbic acid: CB, drug combination
ascorbic acid: DT, drug therapy
carbidopa: CB, drug combination
carbidopa: DT, drug therapy
carbidopa: PR, pharmaceutics
benserazide: CB, drug combination
benserazide: DT, drug therapy
benserazide: PE, pharmacoeconomics
benserazide: PR, pharmaceutics
selegiline: AE, adverse drug reaction
selegiline: IT, drug interaction
dopamine receptor blocking agent: IT, drug interaction
neuroleptic agent: IT, drug interaction
metoclopramide: IT, drug interaction
pergolide: AE, adverse drug reaction
pergolide: DO, drug dose
pergolide: IT, drug interaction
pergolide: DT, drug therapy
pergolide: PE, pharmacoeconomics
pergolide: PD, pharmacology
pramipexole: AE, adverse drug reaction
pramipexole: DO, drug dose
pramipexole: IT, drug interaction
pramipexole: DT, drug therapy
pramipexole: PE, pharmacoeconomics

pramipexole: PK, pharmacokinetics
cimetidine: IT, drug interaction
ranitidine: IT, drug interaction
diltiazem: IT, drug interaction
triamterene: IT, drug interaction
verapamil: IT, drug interaction
quinidine: IT, drug interaction
ropinirole: AE, adverse drug reaction
ropinirole: DO, drug dose
ropinirole: IT, drug interaction
ropinirole: DT, drug therapy
ropinirole: PE, pharmacoeconomics
ropinirole: PK, pharmacokinetics
unindexed drug

RN (iron) 14093-02-8, 53858-86-9, 7439-89-6; (levodopa) 59-92-7; (opiate) 53663-61-9, 8002-76-4, 8008-60-4; (gabapentin) 60142-96-3; (carbamazepine) 298-46-4, 8047-84-5; (clonazepam) 1622-61-3; (caffeine) 30388-07-9, 58-08-2; (alcohol) 64-17-5; (ferritin) 9007-73-2; (tetracycline) 23843-90-5, 60-54-8, 64-75-5; (ascorbic acid) 134-03-2, 15421-15-5, 50-81-7; (carbidopa) 28860-95-9; (benserazide) 14919-77-8, 322-35-0; (selegiline) 14611-51-9, 14611-52-0, 2079-54-1, 2323-36-6; (metoclopramide) 12707-59-4, 2576-84-3, 364-62-5, 7232-21-5; (pergolide) 66104-22-1; (pramipexole) 104632-26-0; (cimetidine) 51481-61-9, 70059-30-2; (ranitidine) 66357-35-5, 66357-59-3; (diltiazem) 33286-22-5, 42399-41-7; (triamterene) 396-01-0; (verapamil) 152-11-4, 52-53-9; (quinidine) 56-54-2; (ropinirole) 91374-21-9

L29 ANSWER 17 OF 27 EMBASE

ACCESSION NUMBER: 2005070684 EMBASE
TITLE: Schizophrenia: A review of neuropharmacology.
AUTHOR: Lyne J.; Kelly B.D.; O'Connor W.T.
CORPORATE SOURCE: Dr. B.D. Kelly, Stanley Research Unit, Department of Adult Psychiatry, Hospitaller Order of St John of God, Newtownpark Avenue, Blackrock, Co Dublin, Ireland. brendankelly35@hotmail.com
SOURCE: Irish Journal of Medical Science, (2004) Vol. 173, No. 3, pp. 155-159. .
Refs: 29
ISSN: 0021-1265 CODEN: IJMSAT
COUNTRY: Ireland
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 030 Pharmacology
032 Psychiatry
037 Drug Literature Index
038 Adverse Reactions Titles
052 Toxicology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 24 Feb 2005
Last Updated on STN: 24 Feb 2005

AB Background. The last few decades have seen significant advances in our understanding of the neurochemical basis of schizophrenia. Aims. To describe the neurotransmitter systems and nerve circuits implicated in schizophrenia; to compare the neuropharmacology of typical and atypical anti-psychotic agents; and to describe recent developments in the pharmacological treatment of schizophrenia. Methods. Relevant pharmacological, neurophysiological and psychiatric literature was examined and reviewed. Results. Schizophrenia is associated with abnormalities of multiple neurotransmitter systems, including dopamine,

serotonin, gamma-aminobutyric acid and glutamate. Typical and atypical antipsychotic agents differ in their receptor-binding affinities, which are related to their differing side-effect profiles. Novel therapeutic strategies include normalisation of synaptic dopamine or serotonin levels, serotonin receptor antagonism and modulation of cerebral protein synthesis. Conclusions. The ideal treatment for schizophrenia may not be a single pharmacological agent but several agents that match the different expressions of the illness, in combination with psycho-social interventions.

CT Medical Descriptors:
*schizophrenia: DT, drug therapy
*schizophrenia: TH, therapy
neuropharmacology
neurotransmission
neurophysiology
drug receptor binding
protein synthesis
synaptic potential
psychosocial care
extrapyramidal symptom: SI, side effect
drug efficacy
dopaminergic activity
negative syndrome: SI, side effect
drug dose regimen
prolactin blood level
hyperprolactinemia: SI, side effect
QT prolongation: SI, side effect
weight gain
  body weight disorder: SI, side effect
sedation
side effect: SI, side effect
drug targeting
drug synthesis
cognition
human
nonhuman
review
Drug Descriptors:
*neuroleptic agent: AE, adverse drug reaction
*neuroleptic agent: CM, drug comparison
*neuroleptic agent: DO, drug dose
*neuroleptic agent: DT, drug therapy
*neuroleptic agent: TO, drug toxicity
*neuroleptic agent: PD, pharmacology
*atypical antipsychotic agent: AE, adverse drug reaction
*atypical antipsychotic agent: CM, drug comparison
*atypical antipsychotic agent: DO, drug dose
*atypical antipsychotic agent: DT, drug therapy
*atypical antipsychotic agent: TO, drug toxicity
*atypical antipsychotic agent: PD, pharmacology
dopamine: EC, endogenous compound
serotonin: EC, endogenous compound
4 aminobutyric acid: EC, endogenous compound
glutamic acid: EC, endogenous compound
serotonin receptor: EC, endogenous compound
brain protein: EC, endogenous compound
chlorpromazine: AE, adverse drug reaction
chlorpromazine: CM, drug comparison
chlorpromazine: DT, drug therapy
chlorpromazine: PD, pharmacology

clozapine: AE, adverse drug reaction
clozapine: CM, drug comparison
clozapine: DO, drug dose
clozapine: DT, drug therapy
clozapine: TO, drug toxicity
clozapine: PD, pharmacology
haloperidol: CM, drug comparison
haloperidol: DO, drug dose
haloperidol: DT, drug therapy
haloperidol: TO, drug toxicity
haloperidol: PD, pharmacology
olanzapine: AE, adverse drug reaction
olanzapine: DO, drug dose
olanzapine: DT, drug therapy
olanzapine: TO, drug toxicity
olanzapine: PD, pharmacology
risperidone: AE, adverse drug reaction
risperidone: DO, drug dose
risperidone: DT, drug therapy
risperidone: TO, drug toxicity
quetiapine: AE, adverse drug reaction
quetiapine: CM, drug comparison
quetiapine: DT, drug therapy
quetiapine: PD, pharmacology
trifluoperazine: CM, drug comparison
trifluoperazine: DT, drug therapy
trifluoperazine: PD, pharmacology
pimozide: CM, drug comparison
pimozide: DT, drug therapy
pimozide: PD, pharmacology
fluphenazine: CM, drug comparison
fluphenazine: DT, drug therapy
fluphenazine: PD, pharmacology
flupentixol: CM, drug comparison
flupentixol: DT, drug therapy
flupentixol: PD, pharmacology
remoxipride: CM, drug comparison
remoxipride: PD, pharmacology
sertindole: AE, adverse drug reaction
sertindole: DT, drug therapy
sertindole: PD, pharmacology
ziprasidone: AE, adverse drug reaction
ziprasidone: DT, drug therapy
ziprasidone: PD, pharmacology
amisulpride: CM, drug comparison
amisulpride: PD, pharmacology
raclopride: CM, drug comparison
raclopride: PD, pharmacology
aripiprazole: AE, adverse drug reaction
aripiprazole: CM, drug comparison
aripiprazole: DT, drug therapy
pramipexole: DV, drug development
serotonin 2A antagonist: DT, drug therapy
serotonin 1A agonist: DT, drug therapy
serotonin 4 agonist: DT, drug therapy
serotonin 4 antagonist: DT, drug therapy
unindexed drug

RN (dopamine) 51-61-6, 62-31-7; (serotonin) 50-67-9; (4 aminobutyric acid) 28805-76-7, 56-12-2; (glutamic acid) 11070-68-1, 138-15-8, 56-86-0, 6899-05-4; (chlorpromazine) 50-53-3, 69-09-0; (clozapine) 5786-21-0;

(haloperidol) 52-86-8; (olanzapine) 132539-06-1; (risperidone) 106266-06-2; (quetiapine) 111974-72-2; (trifluoperazine) 117-89-5, 440-17-5; (pimozide) 2062-78-4; (fluphenazine) 146-56-5, 69-23-8; (flupentixol) 2413-38-9, 2709-56-0; (remoxipride) 78810-02-3, 80125-14-0, 82935-42-0; (sertindole) 106516-24-9; (ziprasidone) 118289-78-4, 122883-93-6, 138982-67-9, 199191-69-0; (amisulpride) 71675-85-9; (raclopride) 84225-95-6; (aripiprazole) 129722-12-9; (pramipexole) 104632-26-0

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ACCESSION NUMBER: 2005139812 EMBASE
TITLE: Restoring energy in a power crisis: Mitochondrial targets for drug development.
AUTHOR: Howell N.; Taylor S.W.; Fahy E.; Murphy A.; Ghosh S.S.
CORPORATE SOURCE: N. Howell, MitoKor Inc., 11494 Sorrento Valley Road, San Diego, CA 92121, United States. howelln@mitokor.com
SOURCE: Drug Discovery Today: TARGETS, (2003) Vol. 2, No. 5, pp. 208-216. .
Refs: 51
ISSN: 1741-8372 CODEN: DDTTA4
PUBLISHER IDENT.: S 1477-3627(03)02364-X
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 008 Neurology and Neurosurgery
018 Cardiovascular Diseases and Cardiovascular Surgery
022 Human Genetics
025 Hematology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 14 Apr 2005
Last Updated on STN: 14 Apr 2005

AB Mitochondria are the organelles responsible for energy production that 'house' many pathways of intermediary metabolism. It should not be surprising, therefore, that several human diseases involve mitochondrial dysfunction or dysregulation, alt hough many of these diseases have complex etiologies that are not yet fully defined. For some of these diseases, there is evidence that ameliorating the mitochondrial dysfunction will provide clinical benefit. Several marketed or late-stage drugs are now known to act on mitochondrial targets, although this was not recognized when they were initially developed. The main requirements for progress in the area of mitochondrial drug development are a more systematic and comprehensive definition of the mitochondrial proteome and the identification of targets for drug development.

CT Medical Descriptors:
*mitochondrion
*disorders of mitochondrial functions: ET, etiology
cell organelle
genome
mitochondrial respiration
gene mutation
bioenergy
MELAS syndrome: ET, etiology
Leber hereditary optic neuropathy: ET, etiology
MERRF syndrome: ET, etiology
NARP syndrome: ET, etiology
Leigh disease: ET, etiology
Friedreich ataxia: ET, etiology

hereditary motor sensory neuropathy: ET, etiology
autosomal dominant optic atrophy: ET, etiology
hearing impairment: ET, etiology
dystonia: ET, etiology
Huntington chorea: ET, etiology
Alzheimer disease: ET, etiology
amyotrophic lateral sclerosis: ET, etiology
progressive supranuclear palsy: ET, etiology
energy yield
disease association
Parkinson disease: ET, etiology
cell inclusion
depression: DT, drug therapy
depression: ET, etiology
  obesity: DT, drug therapy
  obesity: ET, etiology
acute myeloblastic leukemia: DT, drug therapy
acute myeloblastic leukemia: ET, etiology
chronic myeloid leukemia: DT, drug therapy
chronic myeloid leukemia: ET, etiology
breast cancer: DT, drug therapy
breast cancer: ET, etiology
ovary cancer: DT, drug therapy
ovary cancer: ET, etiology
angina pectoris: DT, drug therapy
angina pectoris: ET, etiology
non insulin dependent diabetes mellitus: DT, drug therapy
non insulin dependent diabetes mellitus: ET, etiology
DNA library
DNA sequence
drug research
human
nonhuman
review
Drug Descriptors:
proteome
mitochondrial DNA
mitochondrial protein
huntingtin: EC, endogenous compound
alpha synuclein: EC, endogenous compound
reactive oxygen metabolite: EC, endogenous compound
monoamine oxidase inhibitor: DT, drug therapy
monoamine oxidase inhibitor: PD, pharmacology
adiponectin: DT, drug therapy
adiponectin: PD, pharmacology
arsenic trioxide: DT, drug therapy
arsenic trioxide: PD, pharmacology
paclitaxel: DT, drug therapy
paclitaxel: PD, pharmacology
ranolazine: DT, drug therapy
ranolazine: PD, pharmacology
metformin: DT, drug therapy
metformin: PD, pharmacology
  pramipexole: DT, drug therapy
  pramipexole: PD, pharmacology
cholinesterase inhibitor: DT, drug therapy
anthracycline: DT, drug therapy
anthracycline: PD, pharmacology
cyclosporin A: PD, pharmacology
complementary DNA

famoxin
icorel
RN (huntingtin) 191683-04-2; (alpha synuclein) 154040-18-3; (adiponectin) 283182-39-8; (arsenic trioxide) 1303-24-8, 1327-53-3, 13464-58-9, 15502-74-6; (paclitaxel) 33069-62-4; (ranolazine) 95635-55-5; (metformin) 1115-70-4, 657-24-9; (pramipexole) 104632-26-0; (cyclosporin A) 59865-13-3, 63798-73-2

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ACCESSION NUMBER: 2003213976 EMBASE
TITLE: Ziprasidone-associated mania: A case series and review of the mechanism.
AUTHOR: Baldassano C.F.; Ballas C.; Datto S.M.; Kim D.; Littman L.; O'Reardon J.; Rynn M.A.
CORPORATE SOURCE: Dr. C.F. Baldassano, Hosp. of the Univ. of Pennsylvania, Mood and Anxiety Disorders Clinic, 3535 Market Street, Philadelphia, PA 19104, United States. cfb@mail.med.upenn.edu
SOURCE: Bipolar Disorders, (2003) Vol. 5, No. 1, pp. 72-75. . Refs: 20
ISSN: 1398-5647 CODEN: BDIIAU
COUNTRY: Denmark
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 032 Psychiatry
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 12 Jun 2003
Last Updated on STN: 12 Jun 2003

AB Atypical antipsychotics are now commonly used in the treatment of bipolar disorder, as they have been shown to have effects on mania as well as psychosis. Shortly after the introduction of atypical antipsychotics, several cases of associated hypomania and mania were reported. Ziprasidone is an atypical antipsychotic recently approved by the Food and Drug Administration for the treatment of psychosis. Although ziprasidone has also been shown to be effective in treating mania, it may be associated with the induction of mania or hypomania. We report four cases of mania associated with initiation of ziprasidone, which, to our knowledge, are the first reported for this drug in bipolar patients. As ziprasidone has substantial serotonergic and noradrenergic action, we hypothesize, it may more likely induce mania than other atypical antipsychotics. We advocate future studies to evaluate ziprasidone's efficacy in treating bipolar disorder and caution clinicians that induction of mania or hypomania may be possible with this agent.

CT Medical Descriptors:
*mania: ET, etiology
*mania: SI, side effect
*bipolar disorder: DT, drug therapy
case study
pathophysiology
psychosis: DT, drug therapy
hypomania: SI, side effect
drug approval
food and drug administration
drug efficacy
serotoninergic transmission
noradrenergic system
bipolar I disorder: DT, drug therapy

rapid cycling bipolar disorder: DT, drug therapy
sedation
sleep disorder: SI, side effect
impulsiveness
libido disorder: SI, side effect
distractibility
academic achievement
concentration loss: SI, side effect
euphoria
irritability
thought disorder: SI, side effect
speech disorder: SI, side effect
  body weight disorder: SI, side effect
agitation
angina pectoris: SI, side effect
dose response
human
male
female
case report
adult
review
priority journal
Drug Descriptors:
*ziprasidone: AE, adverse drug reaction
*ziprasidone: CB, drug combination
*ziprasidone: DO, drug dose
*ziprasidone: DT, drug therapy
*ziprasidone: PD, pharmacology
atypical antipsychotic agent: AE, adverse drug reaction
atypical antipsychotic agent: DT, drug therapy
carbamazepine: CB, drug combination
carbamazepine: DT, drug therapy
clonazepam: CB, drug combination
clonazepam: DT, drug therapy
quetiapine: AE, adverse drug reaction
quetiapine: CB, drug combination
quetiapine: DT, drug therapy
serotonin uptake inhibitor: DT, drug therapy
noradrenalin uptake inhibitor: DT, drug therapy
tricyclic antidepressant agent: DT, drug therapy
monoamine oxidase inhibitor: DT, drug therapy
olanzapine: AE, adverse drug reaction
olanzapine: CB, drug combination
olanzapine: DT, drug therapy
risperidone: DT, drug therapy
  pramipexole: DT, drug therapy
tolcapone: CB, drug combination
tolcapone: DT, drug therapy
lithium: CB, drug combination
lithium: DT, drug therapy
lamotrigine: CB, drug combination
lamotrigine: DT, drug therapy
oxcarbazepine: CB, drug combination
oxcarbazepine: DT, drug therapy
perphenazine: DT, drug therapy
valproic acid: CB, drug combination
valproic acid: DT, drug therapy
venlafaxine: CB, drug combination
venlafaxine: DT, drug therapy

RN (ziprasidone) 118289-78-4, 122883-93-6, 138982-67-9, 199191-69-0; (carbamazepine) 298-46-4, 8047-84-5; (clonazepam) 1622-61-3; (quetiapine) 111974-72-2; (olanzapine) 132539-06-1; (risperidone) 106266-06-2; (pramipexole) 104632-26-0; (tolcapone) 134308-13-7; (lithium) 7439-93-2; (lamotrigine) 84057-84-1; (oxcarbazepine) 28721-07-5; (perphenazine) 58-39-9; (valproic acid) 1069-66-5, 99-66-1; (venlafaxine) 93413-69-5

L29 ANSWER 20 OF 27 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2003024794 EMBASE
TITLE: Sleep disorders in a military population.
AUTHOR: Pouliot Z.; Peters M.; Neufeld H.; Delaive K.; Kryger M.H.
CORPORATE SOURCE: Z. Pouliot, Sleep Disorders Center, St. Boniface Gen. Hosp. Res. Center, Winnipeg, Man., Canada
SOURCE: Military Medicine, (1 Jan 2003) Vol. 168, No. 1, pp. 7-10.
.
Refs: 28
ISSN: 0026-4075 CODEN: MMEDA
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 008 Neurology and Neurosurgery
035 Occupational Health and Industrial Medicine
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 29 Jan 2003
Last Updated on STN: 29 Jan 2003

AB Introduction: Sleep disorders are common in the civilian population, but little is known about which sleep disorders are common in members of the military. This article compares a group of military personnel referred to our sleep disorders center with a group of civilian controls also referred to our sleep disorders center. Methods: We analyzed the data of 70 Canadian military personnel and 70 civilian controls matched for age and gender. All subjects had full polysomnography. We compared reasons for referral and final sleep diagnoses for both groups. Results: The mean age of each group was 40.8 ± 7.0 SD (military) and 40.8 ± 7.3 SD (civilians), and there were 61 men and 9 women in each group. Both groups were obese (body mass index, 30.2 ± 5.3 (military) versus 32.5 ± 6.9 (civilian)). Both groups were also pathologically sleepy during the day (Epworth Sleepiness Score, 10.4 ± 4.6 (military) versus 11.3 ± 5.4 (civilian)). The majority of referrals in each group were to rule out a sleep breathing disorder (SBD) (66% military versus 79% civilian, p = not significant). Only military patients were referred to rule out a movement disorder (17.1% military versus 0% civilian; 95% confidence interval of the difference = 8.4%-27.6%, p < 0.05). Fewer military were referred because of excessive daytime sleepiness or insomnia (7.1% military versus 20.0% civilian, 95% confidence interval of the difference = -24.4% to -1.4%, p < 0.05). The most common diagnosis confirmed in both groups was a SBD (53% military, 66% civilian, p = not significant). Conclusions: The range and distribution of sleep disorders seen in the military population is similar to that in the civilian population. Both groups were overweight and sleepy and were found to have SBD and movement disorders. These findings underscore the importance of diagnosing and treating sleep disorders in both groups. The neurocognitive impairment associated with SBD and movement disorders impacts highly on the ability of these groups to safely perform their jobs.
AB Introduction: Sleep disorders are common in the civilian population, but little is known about which sleep disorders are common in members of the

military. This article compares a group of military personnel referred to our sleep disorders center with a group of civilian controls also referred to our sleep disorders center. Methods: We analyzed the data of 70 Canadian military personnel and 70 civilian controls matched for age and gender. All subjects had full polysomnography. We compared reasons for referral and final sleep diagnoses for both groups. Results: The mean age of each group was 40.8 ± 7.0 SD (military) and 40.8 ± 7.3 SD (civilians), and there were 61 men and 9 women in each group. Both groups were obese (body mass index, 30.2 ± 5.3 (military) versus 32.5 ± 6.9 (civilian)). Both groups were also pathologically sleepy during the day (Epworth Sleepiness Score, 10.4 ± 4.6 (military) versus 11.3 ± 5.4 (civilian)). The majority of referrals in each group were to rule out a sleep breathing disorder (SBD) (66% military versus 79% civilian, p = not significant). Only military patients were referred to rule out a movement disorder (17.1% military versus 0% civilian; 95% confidence interval of the difference = 8.4%-27.6%, p < 0.05). Fewer military were referred because of excessive daytime sleepiness or insomnia (7.1% military versus 20.0% civilian, 95% confidence interval of the difference = -24.4% to -1.4%, p < 0.05). The most common diagnosis confirmed in both groups was a SBD (53% military, 66% civilian, p = not significant). Conclusions: The range and distribution of sleep disorders seen in the military population is similar to that in the civilian population. Both groups were overweight and sleepy and were found to have SBD and movement disorders. These findings underscore the importance of diagnosing and treating sleep disorders in both groups. The neurocognitive impairment associated with SBD and movement disorders impacts highly on the ability of these groups to safely perform their jobs.

CT Medical Descriptors:
*sleep disorder: DI, diagnosis
*sleep disorder: DT, drug therapy
*sleep disorder: TH, therapy
Canada
army
polysomnography
patient referral
  obesity
scoring system
motor dysfunction: DI, diagnosis
motor dysfunction: DT, drug therapy
sleep apnea syndrome: DI, diagnosis
sleep apnea syndrome: TH, therapy
insomnia: DI, diagnosis
somnolence
positive end expiratory pressure
human
male
female
major clinical study
controlled study
adult
article
Drug Descriptors:
  pramipexole: DT, drug therapy
RN (pramipexole) 104632-26-0

L29 ANSWER 21 OF 27 MEDLINE on STN
ACCESSION NUMBER: 2002735650 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12499509
TITLE: Gender and pramipexole effects on levodopa

Prepared by: Mary Hale @2-2507 Rem Bldg 1D86

pharmacokinetics and pharmacodynamics.
AUTHOR: Zappia Mario; Quattrone Aldo
SOURCE: Neurology, (2002 Dec 24) Vol. 59, No. 12, pp. 2010; author reply 2010.
Journal code: 0401060. ISSN: 0028-3878.
PUB. COUNTRY: United States
DOCUMENT TYPE: Commentary
Letter
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200301
ENTRY DATE: Entered STN: 27 Dec 2002
Last Updated on STN: 15 Jan 2003
Entered Medline: 14 Jan 2003
TI Gender and pramipexole effects on levodopa pharmacokinetics and pharmacodynamics.
CT Check Tags: Female; Male
*Antiparkinson Agents: AE, adverse effects
*Antiparkinson Agents: PK, pharmacokinetics
Area Under Curve
Biological Availability
Body Weight: PH, physiology
Drug Interactions
Humans
*Levodopa: PK, pharmacokinetics
Sex Characteristics
*Thiazoles: AE, adverse effects

L29 ANSWER 22 OF 27 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2003010176 EMBASE
TITLE: Navigating between scylla and charybdis: Mitochondria are both precedented and novel targets for drug development.
AUTHOR: Howell N.
CORPORATE SOURCE: N. Howell, MitoKor, 11494 Sorrento Valley Road, San Diego, CA 92121, United States. howelln@mitokor.com
SOURCE: Drug Development Research, (1 Oct 2002) Vol. 57, No. 2, pp. 75-82. .
Refs: 70
ISSN: 0272-4391 CODEN: DDREDK
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 016 Cancer
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 16 Jan 2003
Last Updated on STN: 16 Jan 2003
AB Biotechnology companies frequently struggle to convince potential partners or investors that the company's research is cutting-edge, but not so novel as to be an unattractive risk. MitoKor is a biotechnology company that focuses on the development of drugs that act on mitochondrial targets. From a review of the scientific literature, it is clear that a number of drugs act on mitochondrial pathways although that site of action was usually not recognized or understood during early-stage development. At the same time, only a small fraction of all mitochondrial proteins have been identified. Therefore, it appears that mitochondria are a source of both novel and precedented drug targets. With the rapid pace of scientific research, it will be a continuing challenge for biotechnology companies to navigate between being too novel and not being novel enough.

Prepared by: Mary Hale @2-2507 Rem Bldg 1D86

CT Medical Descriptors:
*drug targeting
biotechnology
mitochondrial membrane
apoptosis
target cell
obesity: DT, drug therapy
antineoplastic activity
diabetes mellitus: DT, drug therapy
nerve degeneration
cardiovascular disease: DT, drug therapy
human
article
Drug Descriptors:
*anthracycline antibiotic agent
*lonidamine
*cardiovascular agent: DT, drug therapy
1 ethyl 2 [[3 ethyl 5 (3 methylbenzothiazolin 2 ylidene) 4 oxothiazolidin 2 ylidene]methyl]pyridinium chloride: DV, drug development
benzoporphyrin derivative
curcumin
betulic acid
arsenic trioxide
6 [3 (1 adamantyl) 4 hydroxyphenyl] 2 naphthoic acid: DV, drug development
paclitaxel
pramipexole: DV, drug development
nicorandil
2 (3,4 dihydro 2,2 dimethyl 6 nitro 2h 1,4 benzoxazin 4 yl)pyridine 1 oxide: DV, drug development
metformin: DO, drug dose
metformin: DT, drug therapy
metformin: PD, pharmacology
etomoxir: DV, drug development
selegiline
icorel
RN (lonidamine) 50264-69-2; (1 ethyl 2 [[3 ethyl 5 (3 methylbenzothiazolin 2 ylidene) 4 oxothiazolidin 2 ylidene]methyl]pyridinium chloride) 147366-41-4; (benzoporphyrin derivative) 113719-89-4; (curcumin) 458-37-7; (betulic acid) 472-15-1; (arsenic trioxide) 1303-24-8, 1327-53-3, 13464-58-9, 15502-74-6; (6 [3 (1 adamantyl) 4 hydroxyphenyl] 2 naphthoic acid) 125316-60-1; (paclitaxel) 33069-62-4; (pramipexole) 104632-26-0; (nicorandil) 65141-46-0; (2 (3,4 dihydro 2,2 dimethyl 6 nitro 2h 1,4 benzoxazin 4 yl)pyridine 1 oxide) 136544-11-1; (metformin) 1115-70-4, 657-24-9; (etomoxir) 82258-36-4; (selegiline) 14611-51-9, 14611-52-0, 2079-54-1, 2323-36-6

L29 ANSWER 23 OF 27 EMBASE
ACCESSION NUMBER: 2002184779 EMBASE
TITLE: A preliminary study of the relationship between clozapine-induced weight gain and menstrual irregularities in schizophrenic, schizoaffective, and bipolar women.
AUTHOR: Feldman D.; Goldberg J.F.
CORPORATE SOURCE: Dr. J.F. Goldberg, Payne Whitney Clinic, Box 140, 525 E. 68th Street, New York, NY 10021, United States. jfgoldbe@mail.med.cornell.edu
SOURCE: Annals of Clinical Psychiatry, (2002) Vol. 14, No. 1, pp. 17-21. .
Refs: 18

ISSN: 1040-1237 CODEN: APSYEZ
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 003 Endocrinology
010 Obstetrics and Gynecology
032 Psychiatry
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 6 Jun 2002
Last Updated on STN: 6 Jun 2002

AB Controversy persists about links between psychotropic drug use, obesity, and consequent menstrual irregularities. Although these interrelationships have been suggested to possibly explain polycystic ovarian syndrome among women taking valproate, less is known about menstrual irregularities associated with weight gain caused by other psychotropics. Clozapine, sparing of prolactin-related menstrual effects yet often associated with weight gain, offers a model psychotropic from which to test such hypotheses. We studied outpatient premenopausal women from a clozapine clinic to preliminarily assess the association between menstrual cycle patterns and body mass index (BMI).
Records were reviewed for 13 female premenopausal schizophrenic, bipolar, or schizoaffective outpatients who took clozapine with no conventional antipsychotics for ≥6 months. Mean 6-month menstrual cycle lengths were compared with BMIs and relative weight changes since starting clozapine. Subjects took clozapine (mean ± SD dose 392.2 ± 195.7 mg/day) for a mean ± SD of 4.4 ± 3.2 years, with a mean preclozapine weight increase of 27%. Twenty-three percent had menstrual irregularities in the preceding 6 months (mean ± SD cycle length = 36.4 ± 18.1 days), although no significant associations were observed between cycle length and (a) mean ± SD BMI (32.0 ± 8.4) ($r = -0.09$, $p = 0.78$) or (b) weight change since starting clozapine ($r = -0.10$, $p = 0.75$). The observed lack of association between clozapine -induced weight gain and menstrual disturbances would provisionally suggest that iatrogenic weight gain does not robustly explain the emergence of irregular menses among premenopausal women taking clozapine.
AB Controversy persists about links between psychotropic drug use, obesity, and consequent menstrual irregularities. Although these interrelationships have been suggested to possibly explain polycystic ovarian syndrome among women taking valproate, less is known about menstrual irregularities associated with weight gain caused by other psychotropics. Clozapine, sparing of prolactin-related menstrual effects yet often associated with weight gain, offers a model psychotropic from which to test such hypotheses. We studied outpatient premenopausal women from a clozapine clinic to preliminarily assess the association between menstrual cycle patterns and body mass index (BMI).
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gain does not robustly explain the emergence of irregular menses among premenopausal women taking clozapine.

CT Medical Descriptors:
*schizophrenia: DT, drug therapy
*schizoidism: DT, drug therapy
*manic depressive psychosis: DT, drug therapy
*menstruation disorder: SI, side effect
*obesity: SI, side effect
drug induced disease: SI, side effect
weight gain
body mass
ovary polycystic disease
premenopause
menstrual cycle
pilot study
amenorrhea: SI, side effect
oligomenorrhea: SI, side effect
menorrhagia: SI, side effect
dysmenorrhea: SI, side effect
human
female
clinical article
controlled study
adolescent
adult
article
priority journal
Drug Descriptors:
*clozapine: AE, adverse drug reaction
*clozapine: DO, drug dose
*clozapine: DT, drug therapy
valproic acid
lorazepam
topiramate
amfebutamone
gabapentin
venlafaxine
clonazepam
clomipramine
dexamphetamine
quetiapine
nortriptyline
lamotrigine
pramipexole
sertraline
fluoxetine

RN (clozapine) 5786-21-0; (valproic acid) 1069-66-5, 99-66-1; (lorazepam) 846-49-1; (topiramate) 97240-79-4; (amfebutamone) 31677-93-7, 34911-55-2; (gabapentin) 60142-96-3; (venlafaxine) 93413-69-5; (clonazepam) 1622-61-3; (clomipramine) 17321-77-6, 303-49-1; (dexamphetamine) 1462-73-3, 51-63-8, 51-64-9; (quetiapine) 111974-72-2; (nortriptyline) 72-69-5, 894-71-3; (lamotrigine) 84057-84-1; (pramipexole) 104632-26-0; (sertraline) 79617-96-2; (fluoxetine) 54910-89-3, 56296-78-7, 59333-67-4

L29 ANSWER 24 OF 27 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2002:278870 BIOSIS
DOCUMENT NUMBER: PREV200200278870
TITLE: Restless Legs Syndrome.
AUTHOR(S): Thorp, Micah L. [Reprint author]

Prepared by: Mary Hale @2-2507 Rem Bldg 1D86

CORPORATE SOURCE: Northwest Permanente, 6902 SE Lake Rd., Milwaukie, OR, 97267, USA
Mthorn111@aol.com
SOURCE: International Journal of Artificial Organs, (November, 2001) Vol. 24, No. 11, pp. 755-756. print.
CODEN: IJAODS. ISSN: 0391-3988.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 8 May 2002
Last Updated on STN: 8 May 2002
IT Major Concepts
Methods and Techniques; Neurology (Human Medicine, Medical Sciences); Pharmacology
IT Diseases
diabetes: endocrine disease/pancreas, metabolic disease
Diabetes Mellitus (MeSH)
IT Diseases
insomnia: behavioral and mental disorders, nervous system disease
Sleep Initiation and Maintenance Disorders (MeSH)
IT Diseases
iron deficiency: nutritional disease
Iron: DF, deficiency (MeSH)
IT Diseases
parasthesia: nervous system disease
IT Diseases
restless leg syndrome: nervous system disease, drug therapy, etiology, pathology, therapy, RLS
IT Diseases
rheumatic disease: connective tissue disease, immune system disease, joint disease
Rheumatic Diseases (MeSH)
IT Diseases
uremia: urologic disease
Uremia (MeSH)
IT Chemicals & Biochemicals
D2 subtype dopamine receptors; benzodiazepines: anticonvulsant-drug; clonidine: adrenergic agonist-drug, alpha-adrenergic agonist-drug, antidyskinetic-drug, antihypertensive-drug, autonomic-drug, cardiovascular-drug; gabapentin: analgesic-drug, anticonvulsant-drug; levodopa: antiparkinsonian-drug; opiates: antiparkinsonian-drug; pergolide: antidyskinetic-drug, antiparkinsonian-drug, dopamine receptor agonist-drug; pramipexole: antiparkinsonian-drug; ropinirole: anticonvulsant-drug
RN 12794-10-4 (benzodiazepines)
4205-90-7 (clonidine)
60142-96-3 (gabapentin)
59-92-7 (levodopa)
66104-22-1 (pergolide)
104632-26-0 (pramipexole)
91374-21-9 (ropinirole)

L29 ANSWER 25 OF 27 EMBASE
ACCESSION NUMBER: 1999315270 EMBASE
TITLE: Novel therapeutic strategies.
AUTHOR: Worker C.
CORPORATE SOURCE: C. Worker, Current Drugs Ltd, Middlesex House, 34-42 Cleveland Street, London W1P 6LB, United Kingdom.
charlotte@cursci.co.uk

SOURCE: IDrugs, (1999) Vol. 2, No. 9, pp. 848-852. .
ISSN: 1369-7056 CODEN: IDRUFN
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 037 Drug Literature Index
030 Pharmacology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 30 Sep 1999
Last Updated on STN: 30 Sep 1999

AB Of the many sessions during the first day of the EPHAR meeting, several interesting topics emerged. Among these were a number of presentations investigating novel anti-inflammatory targets, including the search for a selective COX-2 inhibitor and the potential of cytokines/cytokine receptor targets (eg TNFα) as treatments for rheumatoid arthritis (RA) and other chronic inflammatory conditions. Recent advances in the understanding of the pathogenesis of diabetes and obesity have highlighted the need for a multi-therapeutic approach to treatment; several drugs in preclinical investigations were highlighted. Attention was drawn to the potential of AMPA/kainate receptors, historically investigated for the treatment of neurodegenerative disease, which are now showing promise as anti-ischemic therapeutics. Many novel therapeutics strategies were discussed in detail, including the CCK-B antagonists with considerable anxiolytic potential, mitochondrial mechanisms as targets for the treatment of brain injury and the use of stress-activated proteins in anti-ischemic research.
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CT Medical Descriptors:
human
nonhuman
drug mechanism
antiinflammatory activity
rheumatoid arthritis: DT, drug therapy
diabetes mellitus: DT, drug therapy
diabetic obesity: DT, drug therapy
ischemia
brain injury
inflammation
anxiety neurosis: DT, drug therapy
neuroprotection
mitochondrion
mitochondrial respiration
non insulin dependent diabetes mellitus: DT, drug therapy
conference paper
Drug Descriptors:

*prostaglandin synthase: EC, endogenous compound
*cytokine receptor: EC, endogenous compound
*cytokine: EC, endogenous compound
*quisqualic acid receptor: EC, endogenous compound
*kainic acid receptor: EC, endogenous compound
*kainic acid receptor antagonist: DV, drug development
*benzodiazepine derivative: DV, drug development
*cholecystokinin b receptor antagonist: DV, drug development
*anxiolytic agent: DV, drug development
*cyclooxygenase 2 inhibitor: DV, drug development
*AMPA receptor antagonist: DV, drug development
rofecoxib: DV, drug development
celecoxib: DV, drug development
tumor necrosis factor alpha: EC, endogenous compound
infliximab: DV, drug development
etanercept: DV, drug development
insulinotropic peptide: DV, drug development
insulinotropic peptide: PR, pharmaceutics
metformin: PK, pharmacokinetics
acarbose: PK, pharmacokinetics
pramipexole: DV, drug development
chlordiazepoxide: DT, drug therapy
buspirone: DT, drug therapy
gv 15013x: DV, drug development
gv 191869x: DV, drug development
devazepide: DV, drug development
1 (2,3 dihydro 1 methyl 2 oxo 5 phenyl 1h 1,4 benzodiazepin 3 yl) 3 (3 methylphenyl)urea: DV, drug development
n (4 acetyl 1 piperazinyl) 4 fluorobenzamide: DV, drug development
6 quinoxalinecarboxylic acid piperidide: DV, drug development
cyclothiazide: DV, drug development
unindexed drug

RN (prostaglandin synthase) 39391-18-9, 59763-19-8, 9055-65-6; (celecoxib) 169590-42-5; (metformin) 1115-70-4, 657-24-9; (acarbose) 56180-94-0; (pramipexole) 104632-26-0; (chlordiazepoxide) 438-41-5, 58-25-3; (buspirone) 33386-08-2, 36505-84-7; (devazepide) 103420-77-5; (1 (2,3 dihydro 1 methyl 2 oxo 5 phenyl 1h 1,4 benzodiazepin 3 yl) 3 (3 methylphenyl)urea) 118101-09-0; (n (4 acetyl 1 piperazinyl) 4 fluorobenzamide) 133920-70-4; (6 quinoxalinecarboxylic acid piperidide) 154235-83-3; (cyclothiazide) 2259-96-3

L29 ANSWER 26 OF 27 EMBASE

ACCESSION NUMBER: 1999315269 EMBASE
TITLE: EPHAR '99 - Second European Congress of Pharmacology: 3-7 July 1999, Budapest, Hungary.
AUTHOR: Pivac N.; Muck-Seler D.
CORPORATE SOURCE: N. Pivac, Ruder Boskovic Institute, HR-10000 Zagreb, Croatia. Npivac@olimp.irb.hr
SOURCE: IDrugs, (1999) Vol. 2, No. 9, pp. 845-847. .
ISSN: 1369-7056 CODEN: IDRUFN
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 037 Drug Literature Index
030 Pharmacology
008 Neurology and Neurosurgery
032 Psychiatry
018 Cardiovascular Diseases and Cardiovascular Surgery
048 Gastroenterology
LANGUAGE: English

Prepared by: Mary Hale @2-2507 Rem Bldg 1D86

SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 30 Sep 1999
Last Updated on STN: 30 Sep 1999

AB This report represents only a small selection of the various topics discussed at this large meeting. It was characterized by a great deal of information concerning receptor complexity, mechanisms of drug actions and various animal models (for depressions, anxiety/anxiogenic behavior, aggression, knock-out animals, etc). Suprisingly, few new drugs were introduced although some well-known drugs (ie, MAO inhibitors) were introduced for a new use (neuroprotection).

CT Medical Descriptors:
*monoaminergic system
human
nonhuman
rat
knockout mouse
animal model
clinical trial
randomized controlled trial
double blind procedure
multicenter study
drug mechanism
diabetes mellitus: DT, drug therapy
Alzheimer disease: DT, drug therapy
Parkinson disease: DT, drug therapy
heart disease: DT, drug therapy
gastrointestinal disease: DT, drug therapy
brain injury: DT, drug therapy
dose time effect relation
depression: DT, drug therapy
anxiety
aggression
obesity
neuroprotection
conference paper
Drug Descriptors:
*monoamine oxidase inhibitor: PD, pharmacology
*monoamine oxidase inhibitor: DT, drug therapy
*trapidil: PD, pharmacology
*trapidil: DT, drug therapy
*dopamine receptor: EC, endogenous compound
*serotonin uptake inhibitor: PD, pharmacology
*serotonin uptake inhibitor: DT, drug therapy
cgp 3466b: PD, pharmacology
cgp 3466b: DT, drug therapy
selegiline: PD, pharmacology
selegiline: DT, drug therapy
pramipexole: PD, pharmacology
1 (1,4 benzodioxan 5 yl) 4 (2 indanyl)piperazine: PD, pharmacology
n [2 [4 (2 methoxyphenyl) 1 piperazinyl]ethyl] n (2 pyridyl)cyclohexanecarboxamide: PD, pharmacology
dopamine: EC, endogenous compound
serotonin agonist: PD, pharmacology
serotonin receptor: PD, pharmacology
buspirone: PD, pharmacology
gepirone: PD, pharmacology
ipsapirone: PD, pharmacology
tandospirone: PD, pharmacology
rs 30199: PD, pharmacology
fluoxetine: PD, pharmacology

fluoxetine: DT, drug therapy
sibutramine: PD, pharmacology
sibutramine: DT, drug therapy
RN (trapidil) 15421-84-8; (selegiline) 14611-51-9, 14611-52-0, 2079-54-1, 2323-36-6; (pramipexole) 104632-26-0; (1 (1,4 benzodioxan 5 yl) 4 (2 indanyl)piperazine) 146998-34-7; (n [2 [4 (2 methoxyphenyl) 1 piperazinyl]ethyl] n (2 pyridyl)cyclohexanecarboxamide) 146714-97-8; (dopamine) 51-61-6, 62-31-7; (buspirone) 33386-08-2, 36505-84-7; (gepirone) 83928-66-9, 83928-76-1; (ipsapirone) 92589-98-5; (tandospirone) 112457-95-1; (fluoxetine) 54910-89-3, 56296-78-7, 59333-67-4; (sibutramine) 106650-56-0

L29 ANSWER 27 OF 27 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 95175881 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7871089
TITLE: Reversal of stress-induced anhedonia by the dopamine receptor agonist, pramipexole.
AUTHOR: Willner P; Lappas S; Cheeta S; Muscat R
CORPORATE SOURCE: Department of Psychology, City of London Polytechnic, UK.
SOURCE: Psychopharmacology, (1994 Aug) Vol. 115, No. 4, pp. 454-62. Journal code: 7608025. ISSN: 0033-3158.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199503
ENTRY DATE: Entered STN: 7 Apr 1995
Last Updated on STN: 7 Apr 1995
Entered Medline: 28 Mar 1995

AB Chronic exposure to mild unpredictable stress has previously been found to depress the consumption of a palatable (1%) sucrose solution, and to attenuate food-induced place preference conditioning. In this study the effects of pramipexole (SND-919), a dopamine D2 agonist, were studied during 7-9 weeks of chronic treatment. Pramipexole (1.0 mg/kg per day) reversed the suppression of sucrose intake in stressed animals, increasing sucrose intakes above the levels seen in untreated nonstressed controls. Pramipexole also increased sucrose intake in nonstressed animals; these effects were accompanied by increases in water intake and tended to correlate with weight loss. Drug-treated stressed animals also lost weight, but in this case water intake was unaffected. A second group of animals received a higher dose of pramipexole (2.0 mg/kg per day). The effects of the two doses were very similar. After three weeks of treatment, these animals were switched to a lower dose of pramipexole (0.1 mg/kg per day). Increases in sucrose intake were maintained over three weeks of treatment at the lower dose, with significant recovery of body weight. Two further groups received the same doses of pramipexole (1.0 mg/kg for 6 weeks or 2.0 mg/kg for 3 weeks followed by 0.1 mg/kg thereafter), but received intermittent (twice-weekly) drug treatment. Intermittent pramipexole treatments also tended to increase sucrose intakes, but the results were less consistent from week to week. Following 6-8 weeks of pramipexole treatment, food-induced place preference conditioning was studied in all animals.(ABSTRACT TRUNCATED AT 250 WORDS)

TI Reversal of stress-induced anhedonia by the dopamine receptor agonist, pramipexole.

AB Chronic exposure to mild unpredictable stress has previously been found to depress the consumption of a palatable (1%) sucrose solution, and to attenuate food-induced place preference conditioning. In this study the effects of pramipexole (SND-919), a dopamine D2 agonist, were

studied during 7-9 weeks of chronic treatment. Pramipexole (1.0 mg/kg per day) reversed the suppression of sucrose intake in stressed animals, increasing sucrose intakes above the levels seen in untreated nonstressed controls. Pramipexole also increased sucrose intake in nonstressed animals; these effects were accompanied by increases in water intake and tended to correlate with weight loss. Drug-treated stressed animals also lost weight, but in this case water intake was unaffected. A second group of animals received a higher dose of pramipexole (2.0 mg/kg per day). The effects of the two doses were very similar. After three weeks of treatment, these animals were switched to a lower dose of pramipexole (0.1 mg/kg per day). Increases in sucrose intake were maintained over three weeks of treatment at the lower dose, with significant recovery of body weight. Two further groups received the same doses of pramipexole (1.0 mg/kg for 6 weeks or 2.0 mg/kg for 3 weeks followed by 0.1 mg/kg thereafter), but received intermittent (twice-weekly) drug treatment. Intermittent pramipexole treatments also tended to increase sucrose intakes, but the results were less consistent from week to week. Following 6-8 weeks of pramipexole treatment, food-induced place preference conditioning was studied in all animals.(ABSTRACT TRUNCATED AT 250 WORDS)

```
=> s l11 and (children or adolescen? or teenager? or pre(w)teen or puberty or child)
L30          5 FILE MEDLINE
L31          5 FILE BIOSIS
L32         29 FILE EMBASE

TOTAL FOR ALL FILES
L33         39 L11 AND (CHILDREN OR ADOLESCEN? OR TEENAGER? OR PRE(W) TEEN OR
               PUBERTY OR CHILD)

=> s l7 and l33
L34          0 FILE MEDLINE
L35          0 FILE BIOSIS
L36          0 FILE EMBASE

TOTAL FOR ALL FILES
L37          0 L7 AND L33

=> s l33 and l20
L38          0 FILE MEDLINE
L39          0 FILE BIOSIS
L40          1 FILE EMBASE

TOTAL FOR ALL FILES
L41          1 L33 AND L20

=> d ibib abs

L41  ANSWER 1 OF 1  EMBASE  COPYRIGHT (c) 2006 Elsevier B.V. All rights
     reserved on STN
ACCESSION NUMBER:   2002184779  EMBASE
TITLE:              A preliminary study of the relationship between
                    clozapine-induced weight gain and menstrual irregularities
                    in schizophrenic, schizoaffective, and bipolar women.
AUTHOR:             Feldman D.; Goldberg J.F.
CORPORATE SOURCE:   Dr. J.F. Goldberg, Payne Whitney Clinic, Box 140, 525 E.
                    68th Street, New York, NY 10021, United States.
                    jfgoldbe@mail.med.cornell.edu
```

Prepared by: Mary Hale @2-2507 Rem Bldg 1D86

SOURCE: Annals of Clinical Psychiatry, (2002) Vol. 14, No. 1, pp. 17-21. .
Refs: 18
ISSN: 1040-1237 CODEN: APSYEZ
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 003 Endocrinology
010 Obstetrics and Gynecology
032 Psychiatry
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 6 Jun 2002
Last Updated on STN: 6 Jun 2002

AB Controversy persists about links between psychotropic drug use, obesity, and consequent menstrual irregularities. Although these interrelationships have been suggested to possibly explain polycystic ovarian syndrome among women taking valproate, less is known about menstrual irregularities associated with weight gain caused by other psychotropics. Clozapine, sparing of prolactin-related menstrual effects yet often associated with weight gain, offers a model psychotropic from which to test such hypotheses. We studied outpatient premenopausal women from a clozapine clinic to preliminarily assess the association between menstrual cycle patterns and body mass index (BMI).
Records were reviewed for 13 female premenopausal schizophrenic, bipolar, or schizoaffective outpatients who took clozapine with no conventional antipsychotics for ≥6 months. Mean 6-month menstrual cycle lengths were compared with BMIs and relative weight changes since starting clozapine. Subjects took clozapine (mean ± SD dose 392.2 ± 195.7 mg/day) for a mean ± SD of 4.4 ± 3.2 years, with a mean preclozapine weight increase of 27%. Twenty-three percent had menstrual irregularities in the preceding 6 months (mean ± SD cycle length = 36.4 ± 18.1 days), although no significant associations were observed between cycle length and (a) mean ± SD BMI (32.0 ± 8.4) ($r = -0.09$, $p = 0.78$) or (b) weight change since starting clozapine ($r = -0.10$, $p = 0.75$). The observed lack of association between clozapine -induced weight gain and menstrual disturbances would provisionally suggest that iatrogenic weight gain does not robustly explain the emergence of irregular menses among premenopausal women taking clozapine.

```
=> s diabetes mellitus or type(w)(ii or 2) or nody or matur? onset diabetes or
niddm or metabolic syndrome x or glucose metablism disorder
L42      314097 FILE MEDLINE
L43      214134 FILE BIOSIS
L44      259063 FILE EMBASE

TOTAL FOR ALL FILES
L45      787294 DIABETES MELLITUS OR TYPE(W)(II OR 2) OR NODY OR MATUR? ONSET
                DIABETES OR NIDDM OR METABOLIC SYNDROME X OR GLUCOSE METABLISM
               DISORDER

=> s l11 and l45
L46            2 FILE MEDLINE
L47            6 FILE BIOSIS
L48           23 FILE EMBASE

TOTAL FOR ALL FILES
L49           31 L11 AND L45
```

Prepared by: Mary Hale @2-2507 Rem Bldg 1D86

```
=> s l20 and l49
L50          0 FILE MEDLINE
L51          2 FILE BIOSIS
L52          5 FILE EMBASE

TOTAL FOR ALL FILES
L53          7 L20 AND L49

=> dup rem l53
PROCESSING COMPLETED FOR L53
L54          7 DUP REM L53 (0 DUPLICATES REMOVED)

=> d 1-7 ibib abs

L54  ANSWER 1 OF 7  EMBASE  COPYRIGHT (c) 2006 Elsevier B.V. All rights
     reserved on STN
ACCESSION NUMBER:   2005418892  EMBASE
TITLE:              Effective maintenance treatment - Breaking the cycle of
                    bipolar disorder.
AUTHOR:             Goodwin G.; Vieta E.
CORPORATE SOURCE:   G. Goodwin, Department of Psychiatry, Warneford Hospital,
                    University of Oxford, Oxford, United Kingdom.
                    guy.goodwin@psych.ox.ac.uk
SOURCE:             European Psychiatry, (2005) Vol. 20, No. 5-6, pp. 365-371.
                    .
                    Refs: 25
                    ISSN: 0924-9338  CODEN: EUPSED
PUBLISHER IDENT.:   S 0924-9338(05)00120-3
COUNTRY:            France
DOCUMENT TYPE:      Journal; Article
FILE SEGMENT:       032     Psychiatry
                    037     Drug Literature Index
                    038     Adverse Reactions Titles
LANGUAGE:           English
SUMMARY LANGUAGE:   English
ENTRY DATE:         Entered STN: 13 Oct 2005
                    Last Updated on STN: 13 Oct 2005
AB   Clinical guidelines for treatment and research of bipolar disorder greatly
     benefit from the synthesis of data from individual studies.  The British
     Association for Psychopharmacology bases its guidelines on evidence from
     opinions (level D) to systematic reviews of primary trial data (level A).
     The report details conclusions of its 1-day consensus meeting to develop
     guidelines covering diagnosis, clinical management, pharmacotherapy for
     acute episodes, relapse prevention and treatment discontinuation.
     Monotherapy for long-term management is preferred, having reduced
     side-effects and drug interactions and improved compliance.  Combination
     therapy is often preferred for acute episodes, using antipsychotics for
     mania or antidepressants for depression.  Increased efficacy may be
     attributed to multiple mechanisms of action and potentially lower doses.
     In clinical practice, maintenance monotherapy has limited success for
     chronic episodes and polypharmacy is frequently used, though the best
     combination remains unclear.  A new collaborative approach based on simple
     clinical trials is required to change current medical practice. .COPYRGT.
     2005 Elsevier SAS.  All rights reserved.

L54  ANSWER 2 OF 7  BIOSIS  COPYRIGHT (c) 2006 The Thomson Corporation  on STN
ACCESSION NUMBER:   2005:487997  BIOSIS
DOCUMENT NUMBER:    PREV200510290259
TITLE:              Management of augmentatiion in patients with restless legs
```

syndrome.
AUTHOR(S): Trenkwalder, C. [Reprint Author]; Canelo, M.
CORPORATE SOURCE: Paracelsul Elena Klin, Ctr Parkinsonism and Movement Disorders, Kassel, Germany
SOURCE: Sleep (Rochester), (2005) Vol. 28, No. Suppl. S, pp. A278. Meeting Info.: 19th Annual Meeting of the Associated-Professional-Sleep-Societies. Denver, CO, USA. June 18 -23, 2005. Associated Profess Sleep Soc. CODEN: SLEED6. ISSN: 0161-8105.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 16 Nov 2005
Last Updated on STN: 16 Nov 2005

L54 ANSWER 3 OF 7 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2005139812 EMBASE
TITLE: Restoring energy in a power crisis: Mitochondrial targets for drug development.
AUTHOR: Howell N.; Taylor S.W.; Fahy E.; Murphy A.; Ghosh S.S.
CORPORATE SOURCE: N. Howell, MitoKor Inc., 11494 Sorrento Valley Road, San Diego, CA 92121, United States. howelln@mitokor.com
SOURCE: Drug Discovery Today: TARGETS, (2003) Vol. 2, No. 5, pp. 208-216. .
Refs: 51
ISSN: 1741-8372 CODEN: DDTTA4
PUBLISHER IDENT.: S 1477-3627(03)02364-X
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 008 Neurology and Neurosurgery
018 Cardiovascular Diseases and Cardiovascular Surgery
022 Human Genetics
025 Hematology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 14 Apr 2005
Last Updated on STN: 14 Apr 2005
AB Mitochondria are the organelles responsible for energy production that 'house' many pathways of intermediary metabolism. It should not be surprising, therefore, that several human diseases involve mitochondrial dysfunction or dysregulation, alt hough many of these diseases have complex etiologies that are not yet fully defined. For some of these diseases, there is evidence that ameliorating the mitochondrial dysfunction will provide clinical benefit. Several marketed or late-stage drugs are now known to act on mitochondrial targets, although this was not recognized when they were initially developed. The main requirements for progress in the area of mitochondrial drug development are a more systematic and comprehensive definition of the mitochondrial proteome and the identification of targets for drug development. .COPYRGT.2003 Elsevier Science Ltd. All rights reserved.

L54 ANSWER 4 OF 7 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2003010176 EMBASE
TITLE: Navigating between scylla and charybdis: Mitochondria are both precedented and novel targets for drug development.
AUTHOR: Howell N.
CORPORATE SOURCE: N. Howell, MitoKor, 11494 Sorrento Valley Road, San Diego,

CA 92121, United States. howelln@mitokor.com
SOURCE: Drug Development Research, (1 Oct 2002) Vol. 57, No. 2, pp. 75-82. .
Refs: 70
ISSN: 0272-4391 CODEN: DDREDK
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 016 Cancer
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 16 Jan 2003
Last Updated on STN: 16 Jan 2003

AB Biotechnology companies frequently struggle to convince potential partners or investors that the company's research is cutting-edge, but not so novel as to be an unattractive risk. MitoKor is a biotechnology company that focuses on the development of drugs that act on mitochondrial targets. From a review of the scientific literature, it is clear that a number of drugs act on mitochondrial pathways although that site of action was usually not recognized or understood during early-stage development. At the same time, only a small fraction of all mitochondrial proteins have been identified. Therefore, it appears that mitochondria are a source of both novel and precedented drug targets. With the rapid pace of scientific research, it will be a continuing challenge for biotechnology companies to navigate between being too novel and not being novel enough. .COPYRGT. 2002 Wiley-Liss, Inc.

L54 ANSWER 5 OF 7 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2002:278870 BIOSIS
DOCUMENT NUMBER: PREV200200278870
TITLE: Restless Legs Syndrome.
AUTHOR(S): Thorp, Micah L. [Reprint author]
CORPORATE SOURCE: Northwest Permanente, 6902 SE Lake Rd., Milwaukie, OR, 97267, USA
Mthorn111@aol.com
SOURCE: International Journal of Artificial Organs, (November, 2001) Vol. 24, No. 11, pp. 755-756. print.
CODEN: IJAODS. ISSN: 0391-3988.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 8 May 2002
Last Updated on STN: 8 May 2002

L54 ANSWER 6 OF 7 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1999315270 EMBASE
TITLE: Novel therapeutic strategies.
AUTHOR: Worker C.
CORPORATE SOURCE: C. Worker, Current Drugs Ltd, Middlesex House, 34-42 Cleveland Street, London W1P 6LB, United Kingdom.
charlotte@cursci.co.uk
SOURCE: IDrugs, (1999) Vol. 2, No. 9, pp. 848-852. .
ISSN: 1369-7056 CODEN: IDRUFN
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 037 Drug Literature Index
030 Pharmacology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 30 Sep 1999

Prepared by: Mary Hale @2-2507 Rem Bldg 1D86

Last Updated on STN: 30 Sep 1999

AB Of the many sessions during the first day of the EPHAR meeting, several interesting topics emerged. Among these were a number of presentations investigating novel anti-inflammatory targets, including the search for a selective COX-2 inhibitor and the potential of cytokines/cytokine receptor targets (eg TNFα) as treatments for rheumatoid arthritis (RA) and other chronic inflammatory conditions. Recent advances in the understanding of the pathogenesis of diabetes and obesity have highlighted the need for a multi-therapeutic approach to treatment; several drugs in preclinical investigations were highlighted. Attention was drawn to the potential of AMPA/kainate receptors, historically investigated for the treatment of neurodegenerative disease, which are now showing promise as anti-ischemic therapeutics. Many novel therapeutics strategies were discussed in detail, including the CCK-B antagonists with considerable anxiolytic potential, mitochondrial mechanisms as targets for the treatment of brain injury and the use of stress-activated proteins in anti-ischemic research.

L54 ANSWER 7 OF 7 EMBASE

ACCESSION NUMBER: 1999315269 EMBASE
TITLE: EPHAR '99 - Second European Congress of Pharmacology: 3-7 July 1999, Budapest, Hungary.
AUTHOR: Pivac N.; Muck-Seler D.
CORPORATE SOURCE: N. Pivac, Ruder Boskovic Institute, HR-10000 Zagreb, Croatia. Npivac@olimp.irb.hr
SOURCE: IDrugs, (1999) Vol. 2, No. 9, pp. 845-847. .
ISSN: 1369-7056 CODEN: IDRUFN
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 037 Drug Literature Index
030 Pharmacology
008 Neurology and Neurosurgery
032 Psychiatry
018 Cardiovascular Diseases and Cardiovascular Surgery
048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 30 Sep 1999
Last Updated on STN: 30 Sep 1999

AB This report represents only a small selection of the various topics discussed at this large meeting. It was characterized by a great deal of information concerning receptor complexity, mechanisms of drug actions and various animal models (for depressions, anxiety/anxiogenic behavior, aggression, knock-out animals, etc). Suprisingly, few new drugs were introduced although some well-known drugs (ie, MAO inhibitors) were introduced for a new use (neuroprotection).

=> s (food consump? or diet? or eat? behavior) or over eating or food habit or feed? behavior? or food(w)(prefere? or intake) or appetite
L55 394208 FILE MEDLINE
L56 407656 FILE BIOSIS
L57 322737 FILE EMBASE

TOTAL FOR ALL FILES
L58 1124601 (FOOD CONSUMP? OR DIET? OR EAT? BEHAVIOR) OR OVER EATING OR FOOD HABIT OR FEED? BEHAVIOR? OR FOOD(W)(PREFERE? OR INTAKE) OR APPETITE

Prepared by: Mary Hale @2-2507 Rem Bldg 1D86

```
=> s l11 and l58
L59           2 FILE MEDLINE
L60           3 FILE BIOSIS
L61          43 FILE EMBASE

TOTAL FOR ALL FILES
L62          48 L11 AND L58

=> s l62 and (diabetes mellitus or type(w)(ii or 2) or nody or matur? onset
diabetes or niddm or metabolic syndrome x or glucose metablism disorder)
L63           0 FILE MEDLINE
L64           0 FILE BIOSIS
L65           0 FILE EMBASE

TOTAL FOR ALL FILES
L66           0 L62 AND (DIABETES MELLITUS OR TYPE(W)(II OR 2) OR NODY OR MATUR?
                 ONSET DIABETES OR NIDDM OR METABOLIC SYNDROME X OR GLUCOSE
                METABLISM DISORDER)


=> fil caplus
COST IN U.S. DOLLARS                          SINCE FILE      TOTAL
                                                 ENTRY    SESSION
FULL ESTIMATED COST                             138.93     168.60

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FILE LAST UPDATED: 2 Aug 2006  (20060802/ED)

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http://www.cas.org/infopolicy.html

=> s l11
           46 L1
          401 ?PRAMIPEXOLE?
L71       412 L1 OR ?PRAMIPEXOLE?

=> s ((food consump? or diet? or eat? behavior) or over eating or food habit or
feed? behavior? or food(w)(prefere? or intake) or appetite) and l71
        355970 FOOD
         76300 FOODS
        377486 FOOD
                 (FOOD OR FOODS)


Prepared by: Mary Hale @2-2507 Rem Bldg 1D86
```

```
        204508 CONSUMP?
          6675 FOOD CONSUMP?
                 (FOOD(W)CONSUMP?)
        630796 DIET?
         19927 EAT?
        974459 BEHAVIOR
         51686 BEHAVIORS
       1005586 BEHAVIOR
                 (BEHAVIOR OR BEHAVIORS)
           647 EAT? BEHAVIOR
                 (EAT?(W)BEHAVIOR)
       1209539 OVER
           392 OVERS
       1209851 OVER
                 (OVER OR OVERS)
         11523 EATING
             1 EATINGS
         11524 EATING
                 (EATING OR EATINGS)
            25 OVER EATING
                 (OVER(W)EATING)
        355970 FOOD
         76300 FOODS
        377486 FOOD
                 (FOOD OR FOODS)
         15095 HABIT
          7306 HABITS
         21557 HABIT
                 (HABIT OR HABITS)
           381 FOOD HABIT
                 (FOOD(W)HABIT)
        476527 FEED?
       1041679 BEHAVIOR?
          4512 FEED? BEHAVIOR?
                 (FEED?(W)BEHAVIOR?)
        355970 FOOD
         76300 FOODS
        377486 FOOD
                 (FOOD OR FOODS)
        165876 PREFERE?
        109923 INTAKE
         14561 INTAKES
        115402 INTAKE
                 (INTAKE OR INTAKES)
         20125 FOOD(W)(PREFERE? OR INTAKE)
         23004 APPETITE
           185 APPETITES
         23098 APPETITE
                 (APPETITE OR APPETITES)
L72         28 ((FOOD CONSUMP? OR DIET? OR EAT? BEHAVIOR) OR OVER EATING OR
               FOOD HABIT OR FEED? BEHAVIOR? OR FOOD(W)(PREFERE? OR INTAKE) OR
              APPETITE) AND L71

=> s l72 and (diabetes mellitus or type(w)(ii or 2) or nody or matur? onset
diabetes or niddm or metabolic syndrome x or glucose metablism disorder)
        113816 DIABETES
         82978 MELLITUS
         82925 DIABETES MELLITUS
                 (DIABETES(W)MELLITUS)
       1700104 TYPE
```

Prepared by: Mary Hale @2-2507 Rem Bldg 1D86

```
        586664 TYPES
       2153731 TYPE
                 (TYPE OR TYPES)
       2093487 II
           933 IIS
       2094018 II
                 (II OR IIS)
       8821423 2
         97848 TYPE(W)(II OR 2)
            19 NODY
             1 NODIES
            20 NODY
                 (NODY OR NODIES)
        191255 MATUR?
        135730 ONSET
          1093 ONSETS
        136552 ONSET
                 (ONSET OR ONSETS)
        113816 DIABETES
           892 MATUR? ONSET DIABETES
                 (MATUR?(W)ONSET(W)DIABETES)
          4871 NIDDM
            22 NIDDMS
          4875 NIDDM
                 (NIDDM OR NIDDMS)
        222608 METABOLIC
            24 METABOLICS
        222627 METABOLIC
                 (METABOLIC OR METABOLICS)
        114675 SYNDROME
         14930 SYNDROMES
        123205 SYNDROME
                 (SYNDROME OR SYNDROMES)
       1528011 X
          2998 METABOLIC SYNDROME X
                 (METABOLIC(W)SYNDROME(W)X)
        405177 GLUCOSE
           771 GLUCOSES
        405329 GLUCOSE
                 (GLUCOSE OR GLUCOSES)
             5 METABLISM
        249587 DISORDER
        183877 DISORDERS
        387433 DISORDER
                 (DISORDER OR DISORDERS)
             0 GLUCOSE METABLISM DISORDER
                 (GLUCOSE(W)METABLISM(W)DISORDER)
L73          5 L72 AND (DIABETES MELLITUS OR TYPE(W)(II OR 2) OR NODY OR MATUR?
                ONSET DIABETES OR NIDDM OR METABOLIC SYNDROME X OR GLUCOSE
               METABLISM DISORDER)

=> d 1-5 ibib abs hitstr

L73  ANSWER 1 OF 5  CAPLUS  COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER:         2006:469836  CAPLUS
DOCUMENT NUMBER:          144:460844
TITLE:                    Dopamine 3 receptor agonist and antagonist treatment
                          of gastrointestinal motility disorders
INVENTOR(S):              Pasricha, Pankaj Jay; Micci, Maria-Adelaide
PATENT ASSIGNEE(S):       The Board of Regents of the University of Texas
```

System, USA
SOURCE: PCT Int. Appl., 23 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006052626	A2	20060518	WO 2005-US39736	20051103
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
US 2006116350	A1	20060601	US 2005-266686	20051103
PRIORITY APPLN. INFO.:			US 2004-624603P P	20041103

AB The invention discloses that systemic activation of dopamine 3 receptor (D3R) significantly delays gastric emptying in rat, suggesting that D3R plays an important role in the regulation of gastric motility. Specific D3R antagonist, nafadotride, was shown to partially reverse the effect of dopamine on gastric emptying. The invention also discloses that D3R agonists and antagonists can be used to treat gastrointestinal motility disorders.

L73 ANSWER 2 OF 5 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:800792 CAPLUS
DOCUMENT NUMBER: 141:271594
TITLE: Pramipexole for reduction of excessive food intake in children
INVENTOR(S): Mierau, Joachim; Reess, Jurgen; Wienrich, Marion
PATENT ASSIGNEE(S): Boehringer Ingelheim Pharma GmbH & Co. KG, Germany
SOURCE: Ger. Offen., 8 pp.
CODEN: GWXXBX
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
DE 10312809	A1	20040930	DE 2003-10312809	20030321
US 2004266794	A1	20041230	US 2004-801286	20040316
CA 2519584	AA	20040930	CA 2004-2519584	20040318
WO 2004082680	A1	20040930	WO 2004-EP2793	20040318
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ,				

BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

EP 1608367 A1 20051228 EP 2004-721477 20040318
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK

PRIORITY APPLN. INFO.: DE 2003-10312809 A 20030321
US 2003-496747P P 20030821
WO 2004-EP2793 W 20040318

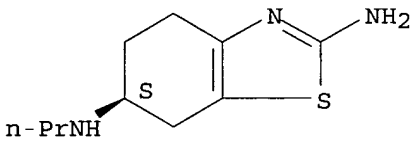
AB The invention discloses the use of dopamine receptor agonists for production of a medicament for reduction of excessive food intake in children.

IT 191217-81-9, Pramipexole dihydrochloride monohydrate
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(pramipexole for reduction of excessive food intake in children)

RN 191217-81-9 CAPLUS

CN 2,6-Benzothiazolediamine, 4,5,6,7-tetrahydro-N6-propyl-, dihydrochloride, monohydrate, (6S)- (9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



● 2 HCl

● H_2O

L73 ANSWER 3 OF 5 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:511137 CAPLUS
DOCUMENT NUMBER: 139:47219
TITLE: Methods of treating fibromyalgia syndrome, chronic fatigue syndrome and pain with dual serotonin-norepinephrine reuptake inhibitor
INVENTOR(S): Rao, Srinivas G.; Kranzler, Jay D.
PATENT ASSIGNEE(S): Cypress Bioscience, Inc., USA
SOURCE: PCT Int. Appl., 75 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 5
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003053426	A1	20030703	WO 2002-US40976	20021219
W: CA, US				

Prepared by: Mary Hale @2-2507 Rem Bldg 1D86

RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR

US 2003130353	A1	20030710	US 2001-28547	20011219
US 6602911	B2	20030805		

PRIORITY APPLN. INFO.: US 2001-28547 A1 20011219
US 2001-14149 A2 20011105

OTHER SOURCE(S): MARPAT 139:47219

AB The present invention provides a method of treating, in a mammal, chronic fatigue syndrome (CFS), chronic fatigue syndrome (CFS) that is associated with depression, a combination of chronic fatigue syndrome (CFS) and fibromyalgia syndrome (FMS), fibromyalgia syndrome (FMS) associated with depression, pain, and pain associated with depression. The method includes administering a therapeutically effective amount of a dual serotonin-norepinephrine reuptake inhibitor compound or a pharmaceutically acceptable salt thereof.

REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L73 ANSWER 4 OF 5 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2003:376684 CAPLUS
DOCUMENT NUMBER: 138:374216
TITLE: Selective norepinephrine serotonin reuptake inhibitors for treating fibromyalgia syndrome, chronic fatigue syndrome and pain
INVENTOR(S): Rao, Srinivas G.; Kranzler, Jay D.
PATENT ASSIGNEE(S): Cypress Bioscience, Inc., USA
SOURCE: PCT Int. Appl., 75 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 5
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003039598	A1	20030515	WO 2002-US35396	20021105
WO 2003039598	C1	20040603		
W: CA, US				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR				
US 2003139476	A1	20030724	US 2001-14149	20011105
US 6635675	B2	20031021		
CA 2467356	AA	20030515	CA 2002-2467356	20021105
EP 1463528	A1	20041006	EP 2002-793880	20021105
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY, TR, BG, CZ, EE, SK				

PRIORITY APPLN. INFO.: US 2001-14149 A 20011105
WO 2002-US35396 W 20021105

OTHER SOURCE(S): MARPAT 138:374216

AB The present invention provides a method of treating, in a mammal, chronic fatigue syndrome (CFS), chronic fatigue syndrome (CFS) that is associated with depression, a combination of chronic fatigue syndrome (CFS) and fibromyalgia syndrome (FMS), fibromyalgia syndrome (FMS) associated with depression, pain and pain associated with depression. The method includes administering a therapeutically effective amount of a dual serotonin norepinepbrine reuptake inhibitor compound or a pharmaceutically acceptable salt thereof. The effect of milnacipran in FMS animal and patients were examined

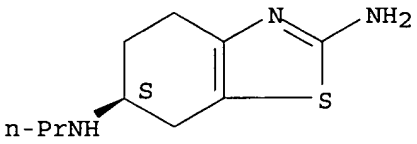
REFERENCE COUNT: 12 THERE ARE 12 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L73 ANSWER 5 OF 5 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:282382 CAPLUS
DOCUMENT NUMBER: 138:292796
TITLE: Dopamine receptor agonists for reducing excessive intake of food
INVENTOR(S): Pieper, Michael Paul; Mierau, Joachim
PATENT ASSIGNEE(S): Boehringer Ingelheim Pharma Kg, Germany
SOURCE: PCT Int. Appl., 12 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003028710	A2	20030410	WO 2002-EP10805	20020926
WO 2003028710	A3	20030912		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
DE 10148233	A1	20030410	DE 2001-10148233	20010928
CA 2461586	AA	20030410	CA 2002-2461586	20020926
EP 1438047	A2	20040721	EP 2002-772350	20020926
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK				
JP 2005504110	T2	20050210	JP 2003-532043	20020926
US 2003087941	A1	20030508	US 2002-259118	20020927
US 2005032843	A1	20050210	US 2004-935507	20040907
US 2005032812	A1	20050210	US 2004-935508	20040907
US 2006030607	A1	20060209	US 2005-244806	20051006
PRIORITY APPLN. INFO.:			DE 2001-10148233 A	20010928
			WO 2002-EP10805 W	20020926
			US 2002-259118 B3	20020927
			US 2004-935507 B1	20040907

AB The invention relates to the use of dopamine receptor agonists for the production of a pharmaceutical for reducing excessive intake of food.
IT 191217-81-9, Pramipexole dihydrochloride monohydrate
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(dopamine receptor agonists for reducing excessive intake of food)
RN 191217-81-9 CAPLUS
CN 2,6-Benzothiazolediamine, 4,5,6,7-tetrahydro-N6-propyl-, dihydrochloride, monohydrate, (6S)- (9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



● 2 HCl

● H_2O

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=> s l72 and (children or adolescen? or teenager? or pre(w)teen or puberty or child)
          62554 CHILDREN
             47 CHILDRENS
          62571 CHILDREN
                  (CHILDREN OR CHILDRENS)
           8634 ADOLESCEN?
            407 TEENAGER?
         201807 PRE
            682 PRES
         202167 PRE
                  (PRE OR PRES)
            121 TEEN
             98 TEENS
            217 TEEN
                  (TEEN OR TEENS)
              1 PRE(W)TEEN
           9280 PUBERTY
              2 PUBERTIES
           9280 PUBERTY
                  (PUBERTY OR PUBERTIES)
          37653 CHILD
             95 CHILDS
          62554 CHILDREN
             47 CHILDRENS
          74746 CHILD
                  (CHILD OR CHILDS OR CHILDREN OR CHILDRENS)
L74           1 L72 AND (CHILDREN OR ADOLESCEN? OR TEENAGER? OR PRE(W)TEEN OR
                  PUBERTY OR CHILD)

=> d ibib abs hitstr

L74  ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER:         2004:800792  CAPLUS
DOCUMENT NUMBER:          141:271594
TITLE:                    Pramipexole for reduction of excessive
                          food intake in children
INVENTOR(S):              Mierau, Joachim; Reess, Jurgen; Wienrich, Marion
PATENT ASSIGNEE(S):       Boehringer Ingelheim Pharma GmbH & Co. KG, Germany
SOURCE:                   Ger. Offen., 8 pp.
                          CODEN: GWXXBX
DOCUMENT TYPE:            Patent
```

Prepared by: Mary Hale @2-2507 Rem Bldg 1D86

LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
DE 10312809	A1	20040930	DE 2003-10312809	20030321
US 2004266794	A1	20041230	US 2004-801286	~~20040316~~
CA 2519584	AA	20040930	CA 2004-2519584	20040318
WO 2004082680	A1	20040930	WO 2004-EP2793	20040318
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1608367	A1	20051228	EP 2004-721477	20040318
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK				

PRIORITY APPLN. INFO.:
DE 2003-10312809 A 20030321
US 2003-496747P P 20030821
WO 2004-EP2793 W 20040318

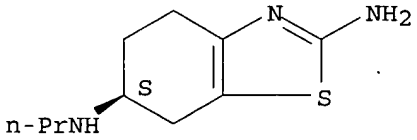
AB The invention discloses the use of dopamine receptor agonists for production of a medicament for reduction of excessive food intake in children.

IT 191217-81-9, Pramipexole dihydrochloride monohydrate
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(pramipexole for reduction of excessive food intake in children)

RN 191217-81-9 CAPLUS

CN 2,6-Benzothiazolediamine, 4,5,6,7-tetrahydro-N6-propyl-, dihydrochloride, monohydrate, (6S)- (9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



● 2 HCl

● H_2O

=> fil medl,biosis,embase,caplus;s mierau j?/au;s reess j?/au;s wienrich m?/au

Prepared by: Mary Hale @2-2507 Rem Bldg 1D86

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	102.36	270.96

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-4.50	-4.50

FILE 'MEDLINE' ENTERED AT 12:42:10 ON 04 AUG 2006

FILE 'BIOSIS' ENTERED AT 12:42:10 ON 04 AUG 2006

FILE 'EMBASE' ENTERED AT 12:42:10 ON 04 AUG 2006

FILE 'CAPLUS' ENTERED AT 12:42:10 ON 04 AUG 2006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L75          20 FILE MEDLINE
L76          41 FILE BIOSIS
L77          24 FILE EMBASE
L78          56 FILE CAPLUS

TOTAL FOR ALL FILES
L79         141 MIERAU J?/AU


L80          11 FILE MEDLINE
L81           7 FILE BIOSIS
L82           8 FILE EMBASE
L83          10 FILE CAPLUS

TOTAL FOR ALL FILES
L84          36 REESS J?/AU


L85          25 FILE MEDLINE
L86          64 FILE BIOSIS
L87          26 FILE EMBASE
L88          40 FILE CAPLUS

TOTAL FOR ALL FILES
L89         155 WIENRICH M?/AU

=> s l79 and l84 and l89
L90           0 FILE MEDLINE
L91           0 FILE BIOSIS
L92           0 FILE EMBASE
L93           1 FILE CAPLUS

TOTAL FOR ALL FILES
L94           1 L79 AND L84 AND L89

=> d ibib abs;s l79 and (l84 or l89);s l84 and l89

L94  ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2006 ACS on STN
```

ACCESSION NUMBER: 2004:800792 CAPLUS
DOCUMENT NUMBER: 141:271594
TITLE: Pramipexole for reduction of excessive food intake in children
INVENTOR(S): Mierau, Joachim; Reess, Jurgen; Wienrich, Marion
PATENT ASSIGNEE(S): Boehringer Ingelheim Pharma GmbH & Co. KG, Germany
SOURCE: Ger. Offen., 8 pp.
CODEN: GWXXBX
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
DE 10312809	A1	20040930	DE 2003-10312809	20030321
US 2004266794	A1	20041230	US 2004-801286	20040316
CA 2519584	AA	20040930	CA 2004-2519584	20040318
WO 2004082680	A1	20040930	WO 2004-EP2793	20040318
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1608367	A1	20051228	EP 2004-721477	20040318
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK				

PRIORITY APPLN. INFO.:

DE 2003-10312809	A	20030321
US 2003-496747P	P	20030821
WO 2004-EP2793	W	20040318

AB The invention discloses the use of dopamine receptor agonists for production of a medicament for reduction of excessive food intake in children.

L95 0 FILE MEDLINE
L96 2 FILE BIOSIS
L97 0 FILE EMBASE
L98 3 FILE CAPLUS

TOTAL FOR ALL FILES
L99 5 L79 AND (L84 OR L89)

L100 0 FILE MEDLINE
L101 0 FILE BIOSIS
L102 0 FILE EMBASE
L103 1 FILE CAPLUS

TOTAL FOR ALL FILES
L104 1 L84 AND L89

=> s l99 or l104

Prepared by: Mary Hale @2-2507 Rem Bldg 1D86

```
L105          0 FILE MEDLINE
L106          2 FILE BIOSIS
L107          0 FILE EMBASE
L108          3 FILE CAPLUS

TOTAL FOR ALL FILES
L109          5 L99 OR L104

=> dup rem l109
PROCESSING COMPLETED FOR L109
L110          5 DUP REM L109 (0 DUPLICATES REMOVED)

=> d 1-5 ibib abs;s (l79 or l84 or l89) and l11

L110 ANSWER 1 OF 5  CAPLUS  COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER:         2005:696747  CAPLUS
DOCUMENT NUMBER:          143:179631
TITLE:                    Pharmaceutical composition comprising a monoamine
                          neurotransmitter re-uptake inhibitor and an
                          N-methyl-D-aspartate (NMDA) receptor antagonist
INVENTOR(S):              Raschig, Andreas; Reess, Juergen; Friedl,
                          Thomas; Mierau, Joachim
PATENT ASSIGNEE(S):       Boehringer Ingelheim International G.m.b.H., Germany;
                          Boehringer Ingelheim Pharma G.m.b.H. & Co. K.-G.
SOURCE:                   PCT Int. Appl., 28 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.           KIND   DATE        APPLICATION NO.         DATE
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     WO 2005070429         A1    20050804    WO 2005-EP167           20050111
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
             CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
             GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,
             LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,
             NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,
             TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
         RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM,
             AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK,
             EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT,
             RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML,
             MR, NE, SN, TD, TG
     US 2005182089         A1    20050818    US 2005-39990           20050121
PRIORITY APPLN. INFO.:                     EP 2004-1283         A   20040122
                                           EP 2004-5818         A   20040311

OTHER SOURCE(S):          MARPAT 143:179631
AB   The invention relates to a pharmaceutical composition comprising a monoamine
     neurotransmitter re-uptake inhibitor comprising a 2,3-disubstituted
     tropane moiety, or a tautomer, a pharmaceutically acceptable salt,
     solvate, or physiol. functional derivative thereof (1), and at least one
     antagonist of N-methyl-D-aspartate (NMDA) receptors or a pharmaceutically
     acceptable salt, solvate, or physiol. functional derivative thereof (2), and a
     pharmaceutically acceptable carrier or excipient, and optionally one or
     more other therapeutic ingredients.
REFERENCE COUNT:          4     THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS
                                RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT
```

L110 ANSWER 2 OF 5 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:395100 CAPLUS
DOCUMENT NUMBER: 142:435801
TITLE: Pharmaceuticals comprising a monoamine neurotransmitter re-uptake inhibitor and an acetylcholinesterase inhibitor
INVENTOR(S): Friedl, Thomas; Mierau, Joachim; Raschig, Andreas; Reess, Juergen; Scheel-Krueger, Joergen
PATENT ASSIGNEE(S): Boehringer Ingelheim International GmbH, Germany; Boehringer Ingelheim Pharma GmbH & Co. Kg; Neurosearch A/S
SOURCE: PCT Int. Appl., 34 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
WO 2005039580	A1	20050506	WO 2004-EP11093		20041005
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW					
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG					
AU 2004283425	A1	20050506	AU 2004-283425		20041005
CA 2542442	AA	20050506	CA 2004-2542442		20041005
EP 1675591	A1	20060705	EP 2004-790120		20041005
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK					
US 2005154009	A1	20050714	US 2004-965994		20041015
PRIORITY APPLN. INFO.:			EP 2003-23635	A	20031016
			EP 2004-5819	A	20040311
			WO 2004-EP11093	W	20041005

OTHER SOURCE(S): MARPAT 142:435801
AB The invention relates to a pharmaceutical composition comprising a monoamine neurotransmitter re-uptake inhibitor comprising a 2,3-disubstituted tropane moiety, or a tautomer, a salt, solvate, or a derivative thereof, and at least one acetylcholinesterase inhibitor and a carrier or excipient, and optionally one or more other therapeutic ingredients. Thus, granules contained a monoamine neurotransmitter re-uptake inhibitor 1.585, rivastigmine hydrogen tartrate 9.597, microcryst. cellulose 66.472, dibasic calcium phosphate 66.471, Hypromellose 2.750, crosslinked CM-cellulose sodiumm 2.000, colloidal silica 0.375, and Mg stearate 0.750 mg/cpsule.
REFERENCE COUNT: 12 THERE ARE 12 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L110 ANSWER 3 OF 5 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:800792 CAPLUS
DOCUMENT NUMBER: 141:271594
TITLE: Pramipexole for reduction of excessive food intake in

children
INVENTOR(S): Mierau, Joachim; Reess, Jurgen; Wienrich, Marion
PATENT ASSIGNEE(S): Boehringer Ingelheim Pharma GmbH & Co. KG, Germany
SOURCE: Ger. Offen., 8 pp.
CODEN: GWXXBX
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
DE 10312809	A1	20040930	DE 2003-10312809	20030321
US 2004266794	A1	20041230	US 2004-801286	20040316
CA 2519584	AA	20040930	CA 2004-2519584	20040318
WO 2004082680	A1	20040930	WO 2004-EP2793	20040318
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1608367	A1	20051228	EP 2004-721477	20040318
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK				

PRIORITY APPLN. INFO.:

DE 2003-10312809	A	20030321
US 2003-496747P	P	20030821
WO 2004-EP2793	W	20040318

AB The invention discloses the use of dopamine receptor agonists for production of a medicament for reduction of excessive food intake in children.

L110 ANSWER 4 OF 5 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 1996:494042 BIOSIS
DOCUMENT NUMBER: PREV199699216398
TITLE: BIIP 20 XX: A potent and selective A1 adenosine receptor antagonist for the treatment of cognitive deficits.
AUTHOR(S): Ensinger, H. A.; Bechtel, W. D.; Gaida, W.; Mierau, J.; Kuefner-Muehl, U.; Wienrich, M.
CORPORATE SOURCE: Dep. Biological Res., 55216 Ingelheim, Germany
SOURCE: Society for Neuroscience Abstracts, (1996) Vol. 22, No. 1-3, pp. 205.
Meeting Info.: 26th Annual Meeting of the Society for Neuroscience. Washington, D.C., USA. November 16-21, 1996.
ISSN: 0190-5295.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 4 Nov 1996
Last Updated on STN: 5 Nov 1996

L110 ANSWER 5 OF 5 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 1994:281103 BIOSIS
DOCUMENT NUMBER: PREV199497294103

TITLE: Effects of selective adenosine A-1 receptor antagonists on synaptic transmission and neuronal activity in rat hippocampal slices.
AUTHOR(S): Gaida, W.; Kuefner-Muehl, U.; Bechtel, W. D.; Mierau, J.; Ensinger, H. A.; Wienrich, M.
CORPORATE SOURCE: Boehringer Ingelheim KG, D-55216 Ingelheim/Rhein, Germany
SOURCE: Naunyn-Schmiedeberg's Archives of Pharmacology, (1994) Vol. 349, No. SUPPL., pp. R96.
Meeting Info.: 35th Spring Meeting of the German Society for Experimental and Clinical Pharmacology and Toxicology. Mainz, Germany. March 15-17, 1994.
CODEN: NSAPCC. ISSN: 0028-1298.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 30 Jun 1994
Last Updated on STN: 18 Nov 1994

L111 5 FILE MEDLINE
L112 8 FILE BIOSIS
L113 11 FILE EMBASE
L114 15 FILE CAPLUS

TOTAL FOR ALL FILES
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L116 0 FILE MEDLINE
L117 0 FILE BIOSIS
L118 1 FILE EMBASE
L119 5 FILE CAPLUS

TOTAL FOR ALL FILES
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PROCESSING COMPLETED FOR L120
L121 6 DUP REM L120 (0 DUPLICATES REMOVED)

=> d 1-6 ibib abs hitstr

L121 ANSWER 1 OF 6 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:696746 CAPLUS
DOCUMENT NUMBER: 143:179630
TITLE: Pharmaceutical composition comprising a monoamine neurotransmitter re-uptake inhibitor and a dopamine agonist
INVENTOR(S): Mierau, Joachim; Pieper, Michael P.
PATENT ASSIGNEE(S): Boehringer Ingelheim International G.m.b.H., Germany; Boehringer Ingelheim Pharma G.m.b.H. & Co. K.-G.
SOURCE: PCT Int. Appl., 30 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005070428	A1	20050804	WO 2005-EP166	20050111
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2005182090	A1	20050818	US 2005-40559	20050121
PRIORITY APPLN. INFO.:			EP 2004-1281 A	20040122
			EP 2004-5817 A	20040311

OTHER SOURCE(S): MARPAT 143:179630

AB The invention relates to a pharmaceutical composition comprising a monoamine neurotransmitter re-uptake inhibitor comprising a 2,3-disubstituted tropane moiety, or a tautomer, a pharmaceutically acceptable salt, solvate, or physiol. functional derivative thereof (1), and at least one dopamine agonist or a pharmaceutically acceptable salt, solvate, or physiol. functional derivative thereof (2), and a pharmaceutically acceptable carrier or excipient, and optionally one or more other therapeutic ingredients.

REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L121 ANSWER 2 OF 6 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:696745 CAPLUS
DOCUMENT NUMBER: 143:199853
TITLE: Monoamine neurotransmitter re-uptake inhibitor comprising a 2,3-disubstituted tropane moiety for the sustained reduction of body weight
INVENTOR(S): Reess, Juergen; Raschig, Andreas; Pollentier, Stephane; Graff, Ole; Mikkelsen, Birgit Ohrt; Priskorn, Morten
PATENT ASSIGNEE(S): Boehringer Ingelheim International G.m.b.H., Germany; Boehringer Ingelheim Pharma G.m.b.H. & Co. K.-G.; Neurosearch A/S
SOURCE: PCT Int. Appl., 35 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005070427	A1	20050804	WO 2005-EP165	20050111
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
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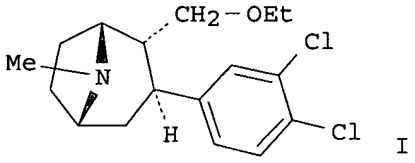
AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2005203124 A1 20050915 US 2005-39991 20050121
PRIORITY APPLN. INFO.: EP 2004-1282 A 20040122
EP 2004-5816 A 20040311

OTHER SOURCE(S): MARPAT 143:199853
GI



I

AB The invention relates to the use of a monoamine neurotransmitter re-uptake inhibitor comprising a 2,3-disubstituted tropane moiety, or a tautomer, a pharmaceutically acceptable salt, solvate, or physiol. functional derivative thereof for the manufacture of a medicament for the sustained reduction of body weight Thus, a tablet was prepared containing a tropane derivative (I) mg, mannitol 121.50 mg, maize starch 79.85 mg, highly dispersed anhydrous silicon dioxide 2.30 mg, Polyvidon K25 2.35 mg, magnesium stearate 3 mg.

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L121 ANSWER 3 OF 6 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:800792 CAPLUS
DOCUMENT NUMBER: 141:271594
TITLE: Pramipexole for reduction of excessive food intake in children
INVENTOR(S): Mierau, Joachim; Reess, Jurgen; Wienrich, Marion
PATENT ASSIGNEE(S): Boehringer Ingelheim Pharma GmbH & Co. KG, Germany
SOURCE: Ger. Offen., 8 pp.
CODEN: GWXXBX
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
DE 10312809	A1	20040930	DE 2003-10312809	20030321
US 2004266794	A1	20041230	US 2004-801286	20040316
CA 2519584	AA	20040930	CA 2004-2519584	20040318
WO 2004082680	A1	20040930	WO 2004-EP2793	20040318

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE,

ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

EP 1608367 A1 20051228 EP 2004-721477 20040318

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK

PRIORITY APPLN. INFO.: DE 2003-10312809 A 20030321
US 2003-496747P P 20030821
WO 2004-EP2793 W 20040318

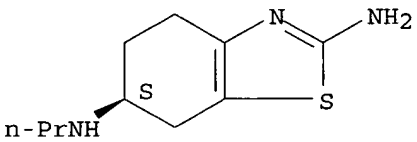
AB The invention discloses the use of dopamine receptor agonists for production of a medicament for reduction of excessive food intake in children.

IT 191217-81-9, Pramipexole dihydrochloride monohydrate
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(pramipexole for reduction of excessive food intake in children)

RN 191217-81-9 CAPLUS

CN 2,6-Benzothiazolediamine, 4,5,6,7-tetrahydro-N6-propyl-, dihydrochloride, monohydrate, (6S)- (9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



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● H_2O

L121 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2003:282382 CAPLUS

DOCUMENT NUMBER: 138:292796

TITLE: Dopamine receptor agonists for reducing excessive intake of food

INVENTOR(S): Pieper, Michael Paul; Mierau, Joachim

PATENT ASSIGNEE(S): Boehringer Ingelheim Pharma Kg, Germany

SOURCE: PCT Int. Appl., 12 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: German

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003028710	A2	20030410	WO 2002-EP10805	20020926
WO 2003028710	A3	20030912		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,

GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH,
PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ,
UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES,
FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF,
CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

DE 10148233	A1	20030410	DE 2001-10148233	20010928
CA 2461586	AA	20030410	CA 2002-2461586	20020926
EP 1438047	A2	20040721	EP 2002-772350	20020926
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK				
JP 2005504110	T2	20050210	JP 2003-532043	20020926
US 2003087941 ✓	A1	20030508	US 2002-259118	20020927
US 2005032843	A1	20050210	US 2004-935507	20040907
US 2005032812	A1	20050210	US 2004-935508	20040907
US 2006030607	A1	20060209	US 2005-244806	20051006

PRIORITY APPLN. INFO.:

DE 2001-10148233	A	20010928
WO 2002-EP10805	W	20020926
US 2002-259118	B3	20020927
US 2004-935507	B1	20040907

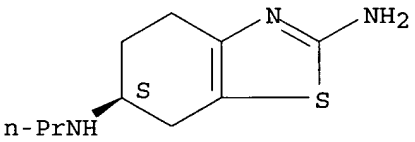
AB The invention relates to the use of dopamine receptor agonists for the production of a pharmaceutical for reducing excessive intake of food.

IT 191217-81-9, Pramipexole dihydrochloride monohydrate
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(dopamine receptor agonists for reducing excessive intake of food)

RN 191217-81-9 CAPLUS

CN 2,6-Benzothiazolediamine, 4,5,6,7-tetrahydro-N6-propyl-, dihydrochloride, monohydrate, (6S)- (9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



● 2 HCl

● H_2O

L121 ANSWER 5 OF 6 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 95273297 EMBASE
DOCUMENT NUMBER: 1995273297
TITLE: Synthesis, pharmacological investigation and computational studies on a tricyclic ergoline analog with selective dopamine autoreceptor activity.
AUTHOR: Gmeiner P.; Bollinger B.; Mierau J.; Hofner G.
CORPORATE SOURCE: Pharmazeutisches Institut, Universitat Bonn, An der

Immenburg 4,D-53121 Bonn, Germany
SOURCE: Archiv der Pharmazie, (1995) Vol. 328, No. 7-8, pp. 609-614. .
ISSN: 0365-6233 CODEN: ARPMAS
COUNTRY: Germany
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 008 Neurology and Neurosurgery
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 10 Oct 1995
Last Updated on STN: 10 Oct 1995

AB The novel aminobenzindolone 8 was prepared and evaluated as a potential antipsychotic agent. The target compound was synthesized in eight steps starting from the tetrahydrobenzindolone 9. The key step of the synthesis was an electrophilic amination of the aromatic ketone 11 followed by reductive degradation when the diethoxymethyl group was employed for protection of the lactam nitrogen and also for the benzylic position 2a. Dopamine and serotonin receptor binding studies revealed 8 to be a potent and selective ligand at the D-2 autoreceptor (k(i) = 4.0 nM). Further in vivo studies including the GBL-test and locomotor activity measurements indicated agonistic activity of 8 at the prejunctional binding sites. Comparison of ab initio based molecular electrostatic isopotential maps corroborates our hypothesis that the dopamine structure 6, containing an intramolecular hydrogen bond donating effect of the meta-HO-group, represents the conformation which is active at the dopamine D-2 autoreceptor.

L121 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1987:5015 CAPLUS
DOCUMENT NUMBER: 106:5015
TITLE: Tetrahydrobenzothiazoles and their use as neurological drugs
INVENTOR(S): Griss, Gerhart; Schneider, Claus; Hurnaus, Rudolf; Kobinger, Walter; Pichler, Ludwig; Bauer, Rudolf; Mierau, Joachim; Hinzen, Dieter; Schingnitz, Guenter
PATENT ASSIGNEE(S): Thomae, Dr. Karl, G.m.b.H., Fed. Rep. Ger.
SOURCE: Eur. Pat. Appl., 57 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
EP 186087	A1	19860702	EP 1985-116016		19851216
EP 186087	B1	19890823			
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DE 3447075	A1	19860703	DE 1984-3447075		19841222
DE 3508947	A1	19860918	DE 1985-3508947		19850313
AT 45735	E	19890915	AT 1985-116016		19851216
PRIORITY APPLN. INFO.:			DE 1984-3447075	A	19841222
			DE 1985-3508947	A	19850313
			EP 1985-116016	A	19851216

OTHER SOURCE(S): CASREACT 106:5015; MARPAT 106:5015
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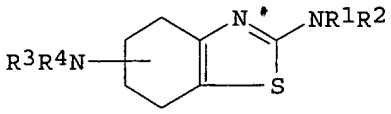


I

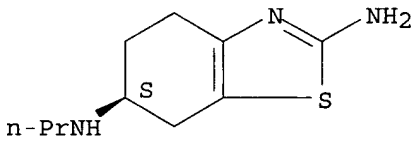
AB Tetrahydrobenzothiazoles I [R1 = H, C1-6 alkyl, C3-6 alkenyl, C3-6 alkynyl, C1-6 alkanoyl, (un)substituted phenylalkyl, phenylalkanoyl; R2 = H, C1-4 alkyl; R3 = H, C1-7 alkyl, C3-7 cycloalkyl, C3-6 alkenyl, C3-6 alkynyl, C1-7 alkanoyl, (un)substituted phenylalkyl, phenylalkanoyl; R4 = H, C1-4 alkyl, C3-6 alkenyl, C3-6 alkynyl; NR3R4 = pyrrolidino, piperidino, hexamethyleneimino, morpholino], their enantiomers and salts, were prepared for the treatment of central nervous diseases and/or circulation problems. Thus, 4-dimethylaminocyclohexanone was brominated and cyclocondensed with H2NCSNH2 to give 2-amino-6-dimethylamino-4,5,6,7-tetrahydrobenzothiazole (II). II inhibited dopamine turnover and parkinsonian syndrome in animal studies. A tablet was formulated containing II 5.0, lactose 33.5, corn starch 10.0, gelatine 1.0, and Mg stearate 0.5 mg.

IT 104632-25-9P
RL: SPN (Synthetic preparation); PREP (Preparation)
(preparation of, as central nervous agent)

RN 104632-25-9 CAPLUS

CN 2,6-Benzothiazolediamine, 4,5,6,7-tetrahydro-N6-propyl-, dihydrochloride, (6S)- (9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



●2 HCl

=> dis his

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FILE 'REGISTRY' ENTERED AT 12:14:54 ON 04 AUG 2006
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L2 3 S E3-E6
L3 3 S L1 OR L2
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E DIABETES TYPE 2/CN 5
E DIABETES MELLITUS TYPE II ?/CN

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E PRAMIPEXOLE/CT 5

Prepared by: Mary Hale @2-2507 Rem Bldg 1D86

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                E DIABETES MELLITUS, TYPE II /CT
                E E3+ALL
                E OBESITY/CT 5
                E E3+ALL
                E EATING DISORDER/CT
                E E3+ALL
                E EATING BEHAVIORS/CT 5
                E E3+ALL
                E GLUT/CT

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L5       168903 FILE BIOSIS
L6       126283 FILE EMBASE
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L8          342 FILE MEDLINE
L9          473 FILE BIOSIS
L10        1592 FILE EMBASE
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L11        2407 S L1 OR ?PRAMIPEXOLE?
L12           1 FILE MEDLINE
L13           2 FILE BIOSIS
L14           6 FILE EMBASE
     TOTAL FOR ALL FILES
L15           9 S L7 AND L11
L16           7 DUP REM L15 (2 DUPLICATES REMOVED)
L17      335586 FILE MEDLINE
L18      411500 FILE BIOSIS
L19      226099 FILE EMBASE
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L21           3 FILE MEDLINE
L22           3 FILE BIOSIS
L23          25 FILE EMBASE
     TOTAL FOR ALL FILES
L24          31 S L20 AND L11
L25           3 FILE MEDLINE
L26           3 FILE BIOSIS
L27          24 FILE EMBASE
     TOTAL FOR ALL FILES
L28          30 S L24 NOT L15
L29          27 DUP REM L28 (3 DUPLICATES REMOVED)
L30           5 FILE MEDLINE
L31           5 FILE BIOSIS
L32          29 FILE EMBASE
     TOTAL FOR ALL FILES
L33          39 S L11 AND (CHILDREN OR ADOLESCEN? OR TEENAGER? OR PRE(W)TEEN OR
L34           0 FILE MEDLINE
L35           0 FILE BIOSIS
L36           0 FILE EMBASE
     TOTAL FOR ALL FILES
L37           0 S L7 AND L33
L38           0 FILE MEDLINE
L39           0 FILE BIOSIS
L40           1 FILE EMBASE
     TOTAL FOR ALL FILES
L41           1 S L33 AND L20
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L42       314097 FILE MEDLINE
L43       214134 FILE BIOSIS
L44       259063 FILE EMBASE
     TOTAL FOR ALL FILES
L45       787294 S DIABETES MELLITUS OR TYPE(W)(II OR 2) OR NODY OR MATUR? ONSET
L46            2 FILE MEDLINE
L47            6 FILE BIOSIS
L48           23 FILE EMBASE
     TOTAL FOR ALL FILES
L49           31 S L11 AND L45
L50            0 FILE MEDLINE
L51            2 FILE BIOSIS
L52            5 FILE EMBASE
     TOTAL FOR ALL FILES
L53            7 S L20 AND L49
L54            7 DUP REM L53 (0 DUPLICATES REMOVED)
L55       394208 FILE MEDLINE
L56       407656 FILE BIOSIS
L57       322737 FILE EMBASE
     TOTAL FOR ALL FILES
L58      1124601 S (FOOD CONSUMP? OR DIET? OR EAT? BEHAVIOR) OR OVER EATING OR F
L59            2 FILE MEDLINE
L60            3 FILE BIOSIS
L61           43 FILE EMBASE
     TOTAL FOR ALL FILES
L62           48 S L11 AND L58
L63            0 FILE MEDLINE
L64            0 FILE BIOSIS
L65            0 FILE EMBASE
     TOTAL FOR ALL FILES
L66            0 S L62 AND (DIABETES MELLITUS OR TYPE(W)(II OR 2) OR NODY OR MAT


     FILE 'CAPLUS' ENTERED AT 12:38:39 ON 04 AUG 2006
L71          412 S L11
L72           28 S ((FOOD CONSUMP? OR DIET? OR EAT? BEHAVIOR) OR OVER EATING OR
L73            5 S L72 AND (DIABETES MELLITUS OR TYPE(W)(II OR 2) OR NODY OR MAT
L74            1 S L72 AND (CHILDREN OR ADOLESCEN? OR TEENAGER? OR PRE(W)TEEN OR

     FILE 'MEDLINE, BIOSIS, EMBASE, CAPLUS' ENTERED AT 12:42:10 ON 04 AUG 2006
L75           20 FILE MEDLINE
L76           41 FILE BIOSIS
L77           24 FILE EMBASE
L78           56 FILE CAPLUS
     TOTAL FOR ALL FILES
L79          141 S MIERAU J?/AU
L80           11 FILE MEDLINE
L81            7 FILE BIOSIS
L82            8 FILE EMBASE
L83           10 FILE CAPLUS
     TOTAL FOR ALL FILES
L84           36 S REESS J?/AU
L85           25 FILE MEDLINE
L86           64 FILE BIOSIS
L87           26 FILE EMBASE
L88           40 FILE CAPLUS
     TOTAL FOR ALL FILES
L89          155 S WIENRICH M?/AU
L90            0 FILE MEDLINE
L91            0 FILE BIOSIS
```

```
L92             0 FILE EMBASE
L93             1 FILE CAPLUS
     TOTAL FOR ALL FILES
L94             1 S L79 AND L84 AND L89
L95             0 FILE MEDLINE
L96             2 FILE BIOSIS
L97             0 FILE EMBASE
L98             3 FILE CAPLUS
     TOTAL FOR ALL FILES
L99             5 S L79 AND (L84 OR L89)
L100            0 FILE MEDLINE
L101            0 FILE BIOSIS
L102            0 FILE EMBASE
L103            1 FILE CAPLUS
     TOTAL FOR ALL FILES
L104            1 S L84 AND L89
L105            0 FILE MEDLINE
L106            2 FILE BIOSIS
L107            0 FILE EMBASE
L108            3 FILE CAPLUS
     TOTAL FOR ALL FILES
L109            5 S L99 OR L104
L110            5 DUP REM L109 (0 DUPLICATES REMOVED)
L111            5 FILE MEDLINE
L112            8 FILE BIOSIS
L113           11 FILE EMBASE
L114           15 FILE CAPLUS
     TOTAL FOR ALL FILES
L115           39 S (L79 OR L84 OR L89) AND L11
L116            0 FILE MEDLINE
L117            0 FILE BIOSIS
L118            1 FILE EMBASE
L119            5 FILE CAPLUS
     TOTAL FOR ALL FILES
L120            6 S ((FOOD CONSUMP? OR DIET? OR EAT? BEHAVIOR) OR OVER EATING OR
L121            6 DUP REM L120 (0 DUPLICATES REMOVED)

=> d l3 que stat;d l3 1-3 ide can
L1              2 SEA FILE=REGISTRY ABB=ON  PLU=ON  PRAMIPEXOLE ?/CN
L2              3 SEA FILE=REGISTRY ABB=ON  PLU=ON  (PRAMIPEXOLE/CN OR "PRAMIPEXO
                  LE DIHYDROCHLORIDE"/CN OR "PRAMIPEXOLE DIHYDROCHLORIDE
                  MONOHYDRATE"/CN OR "PRAMIPEXOLE HYDROCHLORIDE"/CN)
L3              3 SEA FILE=REGISTRY ABB=ON  PLU=ON  L1 OR L2


YOU HAVE REQUESTED DATA FROM FILE 'REGISTRY' - CONTINUE? (Y)/N:n


=> fil reg
COST IN U.S. DOLLARS                            SINCE FILE      TOTAL
                                                   ENTRY    SESSION
FULL ESTIMATED COST                                105.93     376.89

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)      SINCE FILE      TOTAL
                                                   ENTRY    SESSION
CA SUBSCRIBER PRICE                                 -6.75     -11.25

FILE 'REGISTRY' ENTERED AT 12:44:36 ON 04 AUG 2006
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.


Prepared by: Mary Hale @2-2507 Rem Bldg 1D86
```

Property values tagged with IC are from the ZIC/VINITI data file provided by InfoChem.

STRUCTURE FILE UPDATES: 2 AUG 2006 HIGHEST RN 898176-03-9
DICTIONARY FILE UPDATES: 2 AUG 2006 HIGHEST RN 898176-03-9

New CAS Information Use Policies, enter HELP USAGETERMS for details.

TSCA INFORMATION NOW CURRENT THROUGH January 6, 2006

Please note that search-term pricing does apply when conducting SmartSELECT searches.

REGISTRY includes numerically searchable data for experimental and predicted properties as well as tags indicating availability of experimental property data in the original document. For information on property searching in REGISTRY, refer to:

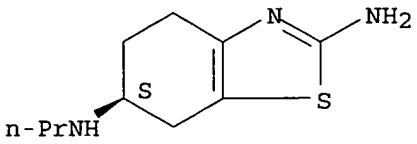
http://www.cas.org/ONLINE/UG/regprops.html

```
=> d l3 que stat;d l3 1-3 ide can
L1            2 SEA FILE=REGISTRY ABB=ON  PLU=ON  PRAMIPEXOLE ?/CN
L2            3 SEA FILE=REGISTRY ABB=ON  PLU=ON  (PRAMIPEXOLE/CN OR "PRAMIPEXO
                LE DIHYDROCHLORIDE"/CN OR "PRAMIPEXOLE DIHYDROCHLORIDE
                MONOHYDRATE"/CN OR "PRAMIPEXOLE HYDROCHLORIDE"/CN)
L3            3 SEA FILE=REGISTRY ABB=ON  PLU=ON  L1 OR L2



L3   ANSWER 1 OF 3  REGISTRY  COPYRIGHT 2006 ACS on STN
RN   191217-81-9  REGISTRY
ED   Entered STN:  16 Jul 1997
CN   2,6-Benzothiazolediamine, 4,5,6,7-tetrahydro-N6-propyl-, dihydrochloride,
     monohydrate, (6S)- (9CI)  (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN   2,6-Benzothiazolediamine, 4,5,6,7-tetrahydro-N6-propyl-, dihydrochloride,
     monohydrate, (S)-
OTHER NAMES:
CN   Mirapex
CN   Pramipexole dihydrochloride monohydrate
FS   STEREOSEARCH
MF   C10 H17 N3 S . 2 Cl H . H2 O
SR   US Adopted Names Council (USAN)
LC   STN Files:   BIOSIS, CA, CAPLUS, IMSPATENTS, IMSRESEARCH, MRCK*,
       PATDPASPC, PS, RTECS*, TOXCENTER, USAN, USPATFULL
         (*File contains numerically searchable property data)
CRN  (104632-26-0)
```

Absolute stereochemistry. Rotation (-).



● 2 HCl

● H_2O

21 REFERENCES IN FILE CA (1907 TO DATE)
22 REFERENCES IN FILE CAPLUS (1907 TO DATE)

REFERENCE 1: 145:826

REFERENCE 2: 144:419741

REFERENCE 3: 144:198965

REFERENCE 4: 143:472478

REFERENCE 5: 143:20045

REFERENCE 6: 142:349474

REFERENCE 7: 142:225816

REFERENCE 8: 141:375099

REFERENCE 9: 141:271594

REFERENCE 10: 141:167814

L3 ANSWER 2 OF 3 REGISTRY COPYRIGHT 2006 ACS on STN
RN 104632-26-0 REGISTRY
ED Entered STN: 11 Oct 1986
CN 2,6-Benzothiazolediamine, 4,5,6,7-tetrahydro-N6-propyl-, (6S)- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN 2,6-Benzothiazolediamine, 4,5,6,7-tetrahydro-N6-propyl-, (S)-
OTHER NAMES:
CN (-)-Pramipexole
CN (S)-2-Amino-6-propylamino-4,5,6,7-tetrahydrobenzothiazole
CN Pramipexole
CN Sifrol
CN SND 919
CN SUD 919CL2Y
CN U 98528E
FS STEREOSEARCH
MF C10 H17 N3 S
CI COM
SR CA
LC STN Files: ADISINSIGHT, ADISNEWS, BEILSTEIN*, BIOSIS, BIOTECHNO, CA,

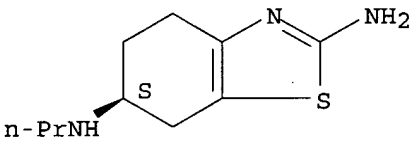
Prepared by: Mary Hale @2-2507 Rem Bldg 1D86

CAPLUS, CASREACT, CBNB, CHEMCATS, CIN, CSCHEM, DDFU, DRUGU, EMBASE, IMSDRUGNEWS, IMSPATENTS, IMSRESEARCH, IPA, MEDLINE, MRCK*, PATDPASPC, PHAR, PROMT, PROUSDDR, PS, RTECS*, SYNTHLINE, TOXCENTER, USAN, USPAT2, USPATFULL

(*File contains numerically searchable property data)

Other Sources: WHO

Absolute stereochemistry. Rotation (-).



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

367 REFERENCES IN FILE CA (1907 TO DATE)
7 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
370 REFERENCES IN FILE CAPLUS (1907 TO DATE)

REFERENCE 1: 145:110313

REFERENCE 2: 145:110

REFERENCE 3: 144:460844

REFERENCE 4: 144:460684

REFERENCE 5: 144:419741

REFERENCE 6: 144:419698

REFERENCE 7: 144:404416

REFERENCE 8: 144:398323

REFERENCE 9: 144:362954

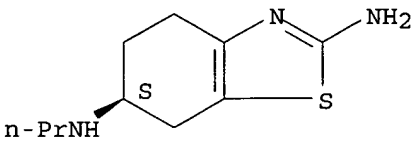
REFERENCE 10: 144:362944

L3 ANSWER 3 OF 3 REGISTRY COPYRIGHT 2006 ACS on STN
RN 104632-25-9 REGISTRY
ED Entered STN: 11 Oct 1986
CN 2,6-Benzothiazolediamine, 4,5,6,7-tetrahydro-N6-propyl-, dihydrochloride, (6S)- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN 2,6-Benzothiazolediamine, 4,5,6,7-tetrahydro-N6-propyl-, dihydrochloride, (S)-
OTHER NAMES:
CN Pramipexole dihydrochloride
CN Pramipexole hydrochloride
CN SND 19
FS STEREOSEARCH
MF C10 H17 N3 S . 2 Cl H
SR CA

Prepared by: Mary Hale @2-2507 Rem Bldg 1D86

LC STN Files: BEILSTEIN*, BIOSIS, CA, CAPLUS, CASREACT, CHEMCATS, IMSPATENTS, IMSRESEARCH, IPA, MRCK*, PATDPASPC, PROUSDDR, PS, RTECS*, SYNTHLINE, TOXCENTER, USPAT2, USPATFULL
(*File contains numerically searchable property data)
CRN (104632-26-0)

Absolute stereochemistry. Rotation (-).



●2 HCl

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

31 REFERENCES IN FILE CA (1907 TO DATE)
32 REFERENCES IN FILE CAPLUS (1907 TO DATE)

REFERENCE 1: 144:419741

REFERENCE 2: 144:331429

REFERENCE 3: 144:192283

REFERENCE 4: 144:128965

REFERENCE 5: 144:128962

REFERENCE 6: 142:225816

REFERENCE 7: 141:370572

REFERENCE 8: 140:416908

REFERENCE 9: 140:151981

REFERENCE 10: 140:151980

=> log y

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	6.86	383.75

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-11.25

STN INTERNATIONAL LOGOFF AT 12:44:43 ON 04 AUG 2006

Prepared by: Mary Hale @2-2507 Rem Bldg 1D86